

CLINICAL ELECTROCARDIOGRAMS

THEIR INTERPRETATION AND SIGNIFICANCE

By

FREDRICK A. WILLIUS, M. D

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PREFACE

In the preparation of this book the author has attempted to present clinical electrocardiography in a graphic manner, and he hopes that the illustrative examples may aid those whose experience in this field is limited. The attempt has been made not only to present typical records of cardiac disorders but also to present records which exhibit transitional changes.

The text of the book has been devoted entirely to the subject of reading of the records themselves and their clinical significance. There has been no attempt to discuss the technic of electrocardiography, the preparation of records, theory, or the many controversial questions. For these data the author refers his readers to the published books and to the innumerable periodical articles on electrocardiography. Since the object of the book is not only to present records and the principal facts regarding their interpretation, but also to aid the reader in pursuing phases in which he is especially interested, a liberal bibliography has been added. The illustrations are accompanied by detailed legends which in themselves comprise a fairly complete text.

The illustrations have been chosen carefully from a large material, and wherever possible the selection has included records the reproduction of which has been clear-cut and striking. In some instances the published records have been reproduced from old tracings which were printed on bromide paper, but these can be identified by symbols in the legends.

It is hoped that this book will be of aid to some, as a reference work, in the interpretation of clinical electrocardiograms.

FREDRICK A. WILLIUS.

The Mayo Clinic
March, 1929.



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CLINICAL ELECTROCARDIOGRAMS

Chapter I

THE NORMAL ELECTROCARDIOGRAM

Electrocardiography was made possible by Einthoven's invention and perfection of the string galvanometer, in 1903. The elaboration of the original instrument, which has occurred with the passage of the years, has given the medical profession the electrocardiograph, an instrument of precision which graphically records the action currents of the heart. During the last few years electrocardiographs have been constructed which utilize the principle of the galvanometer of d'Arsonval combined with recent methods of amplification. Both types of instruments are now generally used in clinical medicine.

Electrocardiography, like other specialized branches of medical science, is frequently evaluated by persons whose acquaintance with the subject is only cursory. The electrocardiogram is the accurate graphic record of the electric activity of the heart, and accumulation of experimental and clinical data has made possible the interpretation and evaluation of many of the deviations from that record which is accepted as the normal one. The graphic records permit exact identification and differentiation of disturbances of cardiac rhythm and of cardiac conduction as well as identification of paroxysms of rapid heart action and of other abnormalities. They do not give reliable diagnostic evidence of valvular lesions nor of diseases of the pericardium. Graphic, clinical, and necropsy correlations have emphasized the importance and reliability of certain abnormalities in prognosis. It is my belief that the science of electrocardiography, even though it is a method of unusual accuracy, should be emphasized in clinical medicine only as an adjunct to other clinical methods of diagnosis. To venture a positive diagnosis of a certain type of lesion from the electrocardiogram alone is an unsound practice, even though in occasional instances this seems to be possible. The trend of modern medicine, regrettably, is to resort too readily to specialized laboratory methods as short-cuts to diagnosis. All of these methods are important contributions to medical science and are records of scientific progress; but the art of physical diagnosis is, unfortunately, being slighted.

It is important for all clinicians who are interested in electrocardiography to correlate the records with clinical data and, when necropsy is available, with the proved pathologic lesions. Continued clinical investigation of this type will result in much more positive knowledge than is available today.

LEADS

Electrocardiographic records may be obtained by applying suitable electrodes to the body at various points. The customary leads adopted, taken in pairs, are from the right arm and the left arm (lead I); from the right arm and the left leg (lead II), and from the left arm and the left leg (lead III). The type and form of the record is dependent

partly on the lead employed, and two leads do not result in identical reproductions. Each lead furnishes data of importance which, under certain conditions, will be found in one lead but will be absent from another.

If electrocardiograms are taken from a patient, simultaneously in leads I and III, with two galvanometers, the algebraic summation of the waves will yield those occurring in lead II. Lead II, then, may be considered a composite lead, for, in general, it is determined by leads I and III. The algebraic summation of leads I and III in ordinary clinical records will not yield lead II, as the records are not obtained simultaneously.

WAVES

The waves or deflections of the electrocardiogram have been designated arbitrarily by the symbols P, Q, R, S, and T (Figs. 1 to 5). The electrocardiogram of man and of

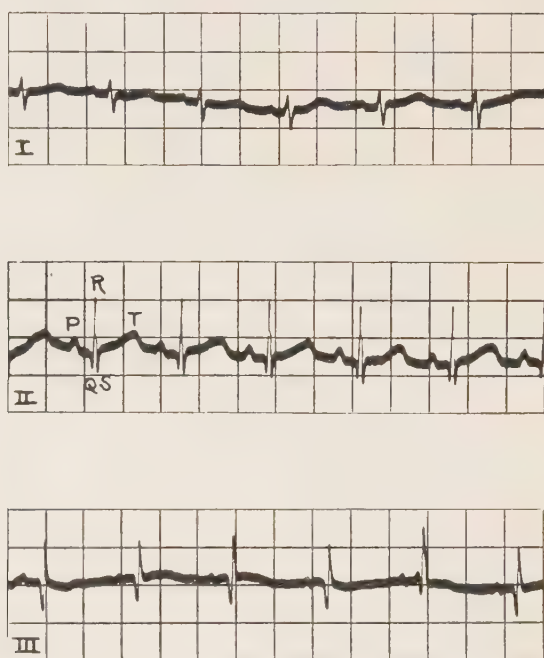


FIG. 1.—Record showing a normal electrocardiogram obtained from a male infant, aged one month.*

other mammals is comprised of an auricular and a ventricular component; the former is represented by the P wave and the latter, by the deflections Q, R, S, and T. The Q and S deflections, when present, are short, abrupt peaks directed downward (negative); they blend with the ascending and the descending limbs of the R wave. The R wave is usually tall and has an amplitude of deflection of from 10 to 15 millivolts. It is a rapid deflection of abrupt contour. The base width of the R wave normally does not exceed 0.1 second and frequently is considerably less.

The T wave follows the QRS complex and is ordinarily a rounded wave with an amplitude of deflection varying from 3 to 7 millivolts. It is preceded and followed by a

* In all the records reproduced in this book the horizontal lines represent current strength: One division = 0.5 millivolt (0.0005 volt). Of the vertical lines which represent the unit of time, each represents 0.2 second. The Roman numeral which appears at the left edge of each record represents the lead employed in making the record.

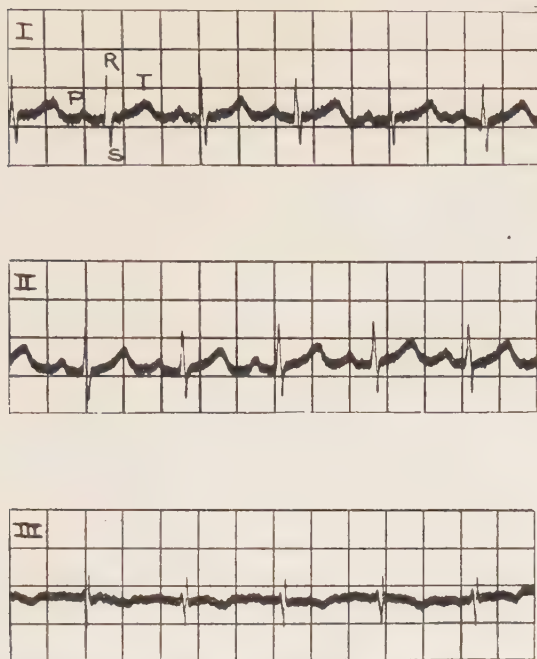


FIG. 2.—Record showing the normal electrocardiogram of a male infant, aged six months. Note the preponderance of the right ventricle.

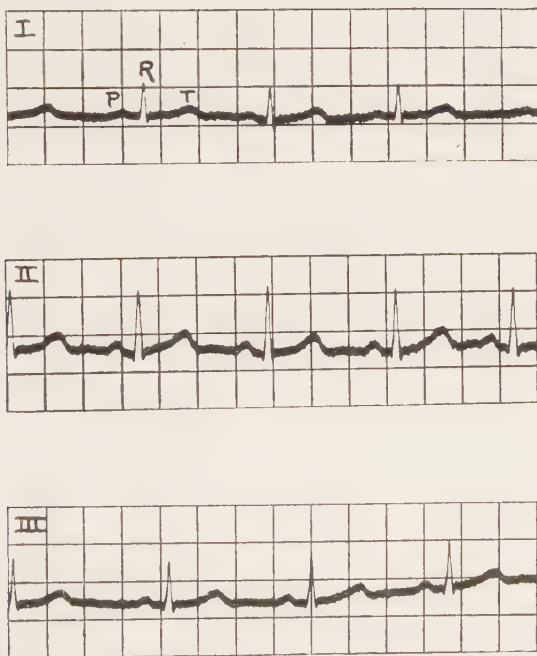


FIG. 3.—Record showing a normal electrocardiogram of a female child in the first decade of life.

horizontal line which, in the various instances, represents iso-electric periods of varying lengths. The T wave occasionally is followed by a small blunt wave which is designated as U.

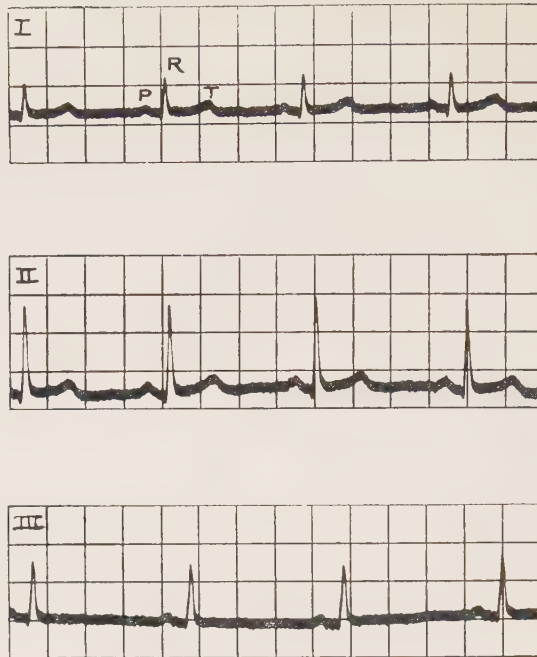


FIG. 4.—Record showing a normal electrocardiogram obtained from a man in the fifth decade of life

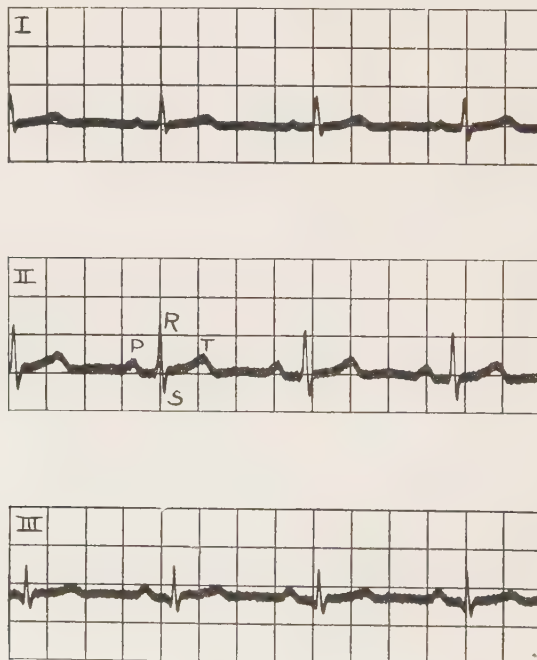


FIG. 5.—Record showing a normal electrocardiogram obtained from a man in the sixth decade of life.

The upstroke of the P wave occurs from 0.13 to 0.21 second before the R wave, and this period constitutes the P-R interval. The S-T interval, which is the distance between the S wave and the termination of the T wave, occupies between 0.24 and 0.28 second.

In most instances the R wave is tallest in lead II, but at times its amplitude may be greatest in lead III. Notching of the P waves not infrequently occurs in normal electrocardiograms, and the QRS complexes in lead III are at times notched.

MEANING OF THE WAVES

As the result of experimental work done by numerous investigators, the meaning of the components of the normal electrocardiogram has been determined in part. I will not attempt to detail the existing evidence for the present conception of the origin of the individual waves, but will refer the reader to the original contributions dealing with the subject and to the current textbooks.

The P wave occurs in relation to the auricular systole and probably also represents the conduction of the impulse through the auricles. The absence of the P wave in auricular fibrillation, when it is known that dynamic contraction has ceased, is important evidence to support this view. The diffusion of the contraction in the auricles is comparatively slow; therefore, when the P wave has been completed, the process of contraction has involved all the auricular tissue.

The course traversed by the wave of excitation over the ventricular muscle has been determined. It enters the special conduction system of the ventricles through the auriculoventricular (His) bundle, passes along the two main divisions of the bundle and spreads through the Purkinje plexus which lines the endocardial surface of both ventricles. From the specialized conduction system the wave of excitation spreads outward through the ventricular muscle toward the epicardial surface of the heart. It has been shown that the rate of travel of a wave of excitation in the conduction system exceeds by tenfold that through ordinary cardiac muscle. The form and direction of the QRS complex depends on the course taken by the wave of excitation through the ventricular muscle, and the duration of the QRS complex depends on the time consumed by the wave of excitation in completing its passage.

As the result of the adequate system of distribution of impulse to the ventricles, about the same time is required for this distribution as is necessary for the dissemination of impulse in the auricles and for their contraction. This is true in spite of the fact that the mass of muscle in the auricles is much smaller than that in the ventricles. The manner of spread of the wave of excitation in the ventricles also results in a constantly changing electropotential at successive instants. Therefore during the inscription of a wave of excitation, in a given lead, the inscription may be directed downward at first, then upward and then downward, as the current within the mass of muscle is first in one direction and then in another.

The QRS complex is followed by a straight line which indicates a fairly uniform equalization of electric effects in all portions of the ventricles.

Much uncertainty still shrouds the meaning of the T wave. It is clearly a record of a ventricular event and perhaps is the expression of relaxation or decline of the state of excitation in the ventricular muscle. Numerous hypotheses regarding this wave have been advanced; yet none of them has received unquestioned confirmation. The T wave is a significant component of the electrocardiogram, as its abnormalities have been definitely correlated with pathologic states.

The meaning of the inconstant summit, U, which at times follows the T wave, is not understood.

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Chapter II

VENTRICULAR PREPONDERANCE

Einthoven was the first to associate certain variations in direction of the initial ventricular complexes with hypertrophy of the left or the right ventricle. He described exaggeration of the R wave in lead I and of the S wave in lead III, as indicative of left ventricular hypertrophy. Conversely, he associated exaggeration of the S wave in lead I, and of the R wave in lead III, with hypertrophy of the right ventricle (Table I and Figs. 6 to 15).

TABLE I

TABULATION OF THE ELECTROCARDIOGRAPHIC SIGNS OF VENTRICULAR PREPONDERANCE*

	Lead I.			Lead III.		
	Q.	R.	S.	Q.	R.	S.
Left ventricular preponderance.	Downward.	Downward.	Upward.	Upward.	Upward.	Downward.
Right ventricular preponderance.	Upward.	Upward.	Downward.	Downward.	Downward.	Upward.

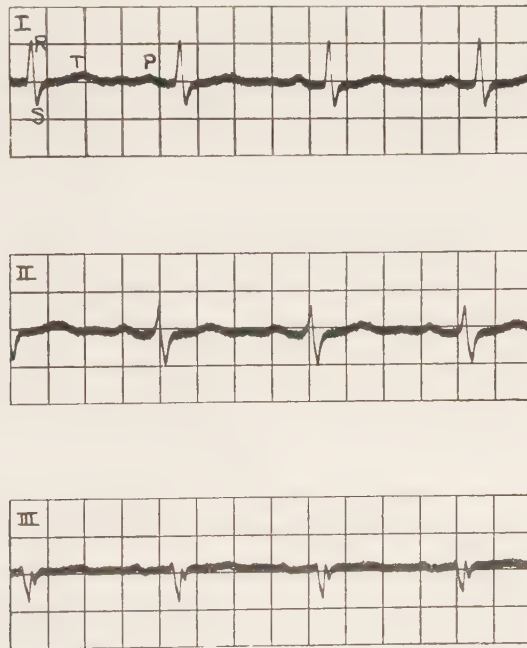


FIG. 6.—Record showing slight preponderance of the right ventricle. Note the downward depression of the S waves in lead I. The QRS complexes in lead III are notched. Record obtained from an obese man aged fifty-two years.

Considerable clinical evidence exists to support these ideas, although I believe that it is erroneous to accept the graphic signs alone in determination of ventricular hyper-

* The words "downward" and "upward" appear beneath the letters which represent the various waves of the QRS component and indicate, in each instance, the direction of the apex in relation to the iso-electric line.

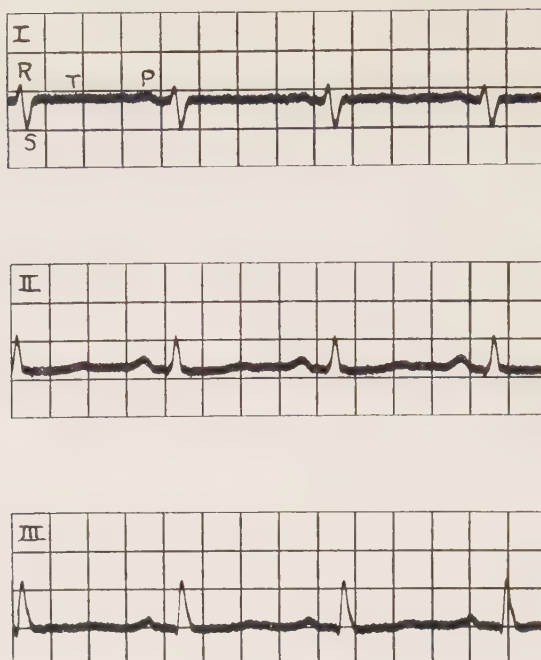


FIG. 7.—Record showing rather slight degree of preponderance of the right ventricle, as evidenced by the prolongation of the S waves in lead I. Record obtained from a man, aged fifty-nine, who had chronic endocarditis with mitral stenosis.

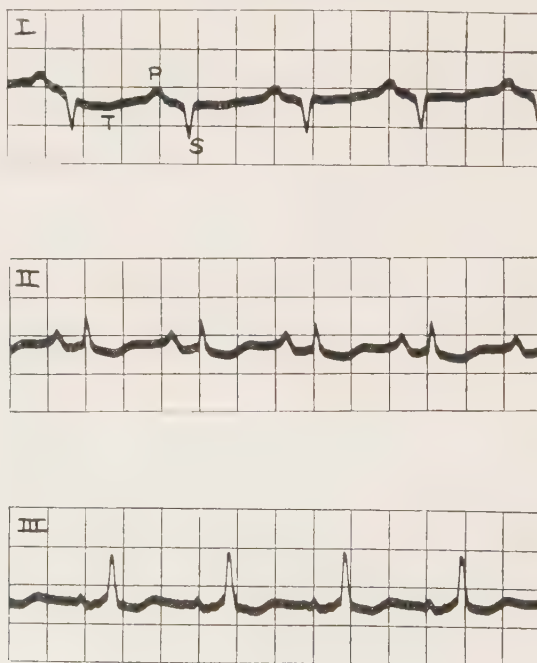


FIG. 8.—Record showing preponderance of the right ventricle as seen by the prolongation of the S waves in lead I. There is an exaggeration in amplitude of the P waves in leads I and II and the T waves are inverted in all leads. Record obtained from a woman, aged thirty-eight years, who had chronic rheumatic endocarditis with mitral stenosis.

trophy. In many cases of hypertension, aortic stenosis, and aortic insufficiency, electrocardiograms are obtained, which display clearly the criteria of Einthoven and which are

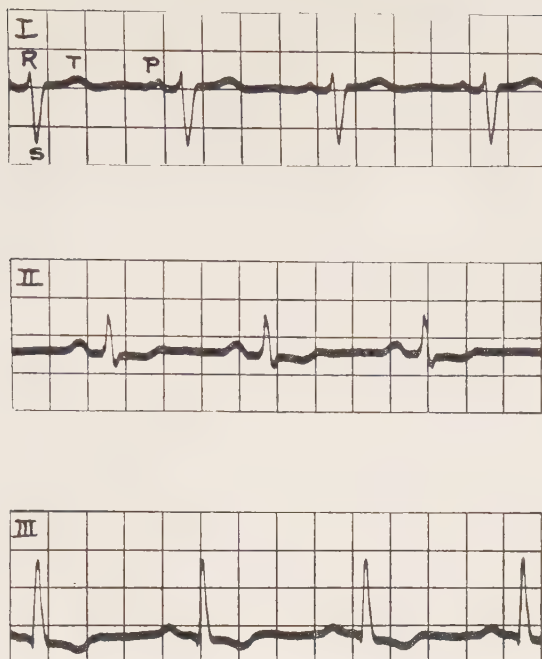


FIG. 9.—Record showing rather marked preponderance of the right ventricle. Note the downward prolongation of the S waves in lead I. The T waves in lead III are inverted. Record obtained from a man, aged thirty-four years, who had chronic rheumatic endocarditis with mitral stenosis.

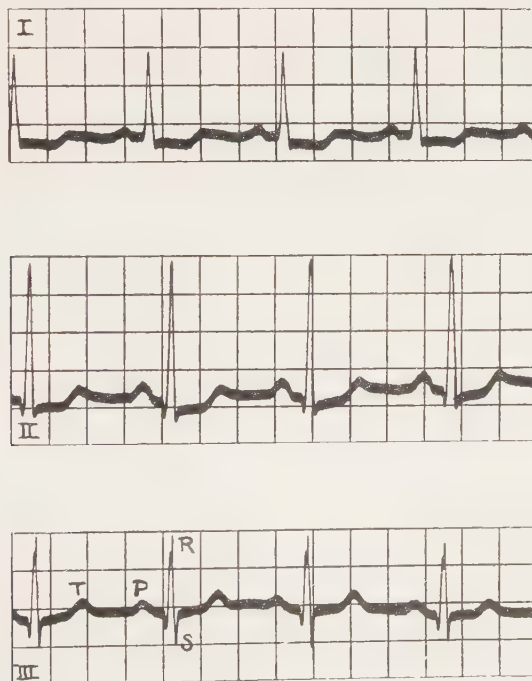


FIG. 10.—Record showing slight preponderance of the left ventricle. Note downward prolongation of the S waves in lead III. The T waves in leads I and II are diphasic. Record obtained from a woman, aged fifty-seven years, with hypertensive heart disease.

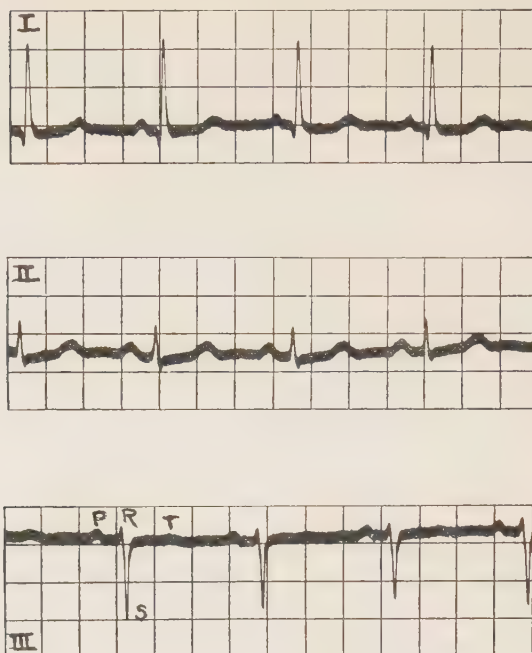


FIG. 11.—Record showing considerable preponderance of the left ventricle as evidenced by the deep downward depression of the S waves in lead III. Record obtained from a woman, aged forty-one years, with exophthalmic goiter.

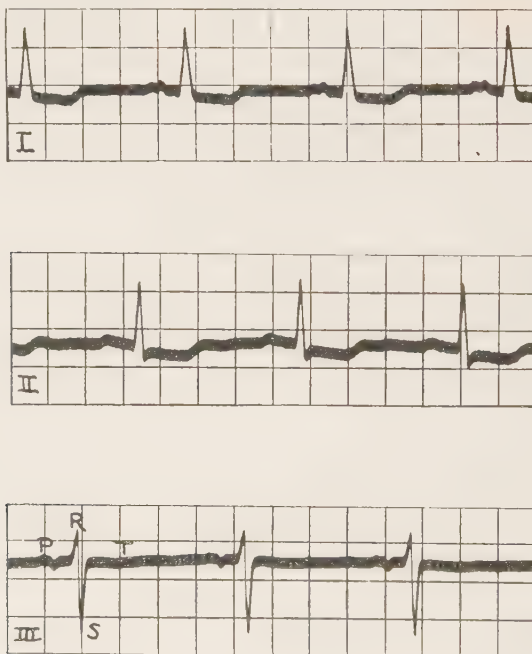


FIG. 12.—Record showing rather marked preponderance of the left ventricle, as evidenced by the deep depression of the S waves in lead III. Note also the T wave inversions in leads I and II and the iso-electric T waves in lead III. The P waves in lead III are diphasic. Record obtained from a man, aged fifty-five years, with hypertensive heart disease.

indicative of left ventricular preponderance. Likewise, many cases of mitral stenosis have been observed in which the graphic signs of right ventricular preponderance are

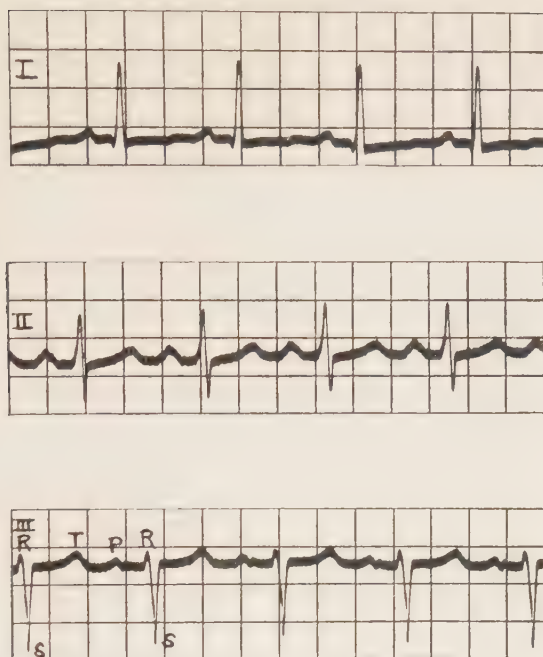


FIG. 13.—Record showing rather marked preponderance of the left ventricle, as evidenced by the deep S depressions in lead III. The T waves in lead I are diphasic. Record obtained from a man, aged forty-two years, with hypertensive heart disease.

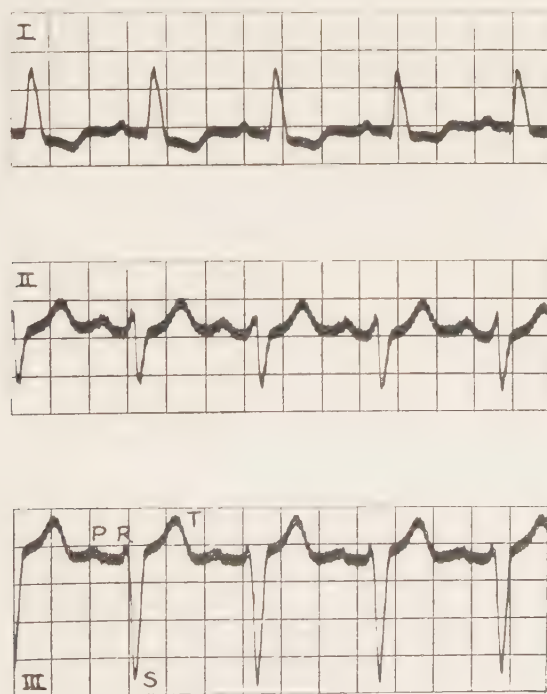


FIG. 14.—Record showing marked preponderance of the left ventricle, as evidenced by the marked downward depression of the S deflections in lead III. Note the T wave inversions in lead I. Record obtained from a man, aged sixty-four years, with hypertensive heart disease.

present. However, in routine clinical electrocardiography many cases are encountered in which clinical and roentgenologic data and graphic signs do not agree. It is not unusual to observe electrocardiograms which exhibit the criteria of right ventricular preponderance but which have been made from patients who have hypertension or aortic disease, with unmistakable clinical evidence of left ventricular hypertrophy. To observe the converse of this is not unusual nor is it uncommon to note the absence of graphic signs of preponderance when clinical evidence clearly indicates hypertrophy of one ventricle or of the other.

It becomes necessary therefore to distinguish clearly between the terms "hypertrophy" and "preponderance." Ventricular preponderance, as displayed by the electrocardiogram, is the expression of the electric effects of one ventricle in relation to the

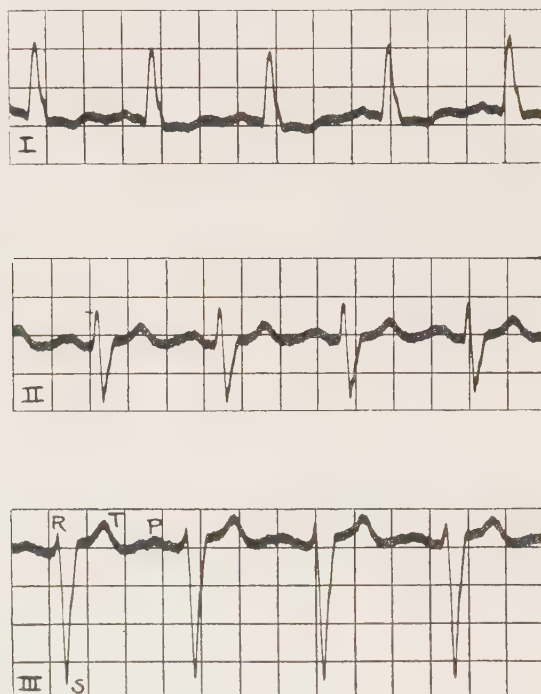


FIG. 15.—Record showing marked preponderance of the left ventricle as evidenced by the deep downward depression of the S waves in lead III. The T waves are inverted in lead I. Record obtained from a man, aged seventy-one years, with coronary sclerosis.

other. The heart may be normal in size and weight, and yet the electric balance of the two ventricles may depart from the normal. The balance between the mass of muscle on the right side and that on the left side may become disturbed from various causes.

Previously it was believed that there was a constant relationship between the separate weight of the two ventricles on one hand and the graphic criteria of ventricular preponderance on the other. This, however, does not appear to be true, except that a more constant agreement is found in greatly hypertrophied hearts. When ventricular weight is below 250 gm., a constant relationship is not evident.

There is a resemblance between the graphic records of preponderance and those which result presumably from defective conduction in the right and the left bundle branches. For instance, the direction of the QRS components, in records which are believed to repre-

sent disturbed conduction in the right bundle branch, is similar to that in electrocardiograms of left ventricular preponderance. Conversely, there is similarity between the graphs of impaired conduction in the left bundle branch and records of right ventricular preponderance. The predominating ventricle clearly determines the inscription of the initial phases of the ventricular electrocardiogram. Other changes, however, occur in the electrocardiograms of bundle-branch block; therefore they are not readily confused with curves of preponderance. These changes will be discussed fully in another chapter.

It has been shown that the position of the heart within the chest influences the electrocardiogram, especially the initial ventricular components. These graphic changes consist of a variation in height and direction of the deflections in all leads.

Changes in position of the normal heart occur chiefly through the movements of the diaphragm. The heart tends to assume a transverse position during full expiration and to assume a vertical position as the diaphragm is lowered during inspiration. As the heart is well fixed at its base, a certain degree of rotation accompanies the excursion of the diaphragm. If fluoroscopic examination is made from in front, it will be found that the direction of rotation during inspiration is clockwise and during expiration, counter-clockwise. The average rotation is about 15 degrees, rarely as much as 30 degrees. Rotation of 25 to 30 degrees definitely would influence all the leads of the electrocardiogram, and the effect would be most pronounced in that lead in which the waves have the least relative value. In persons in whom the diaphragm is normally high a permanent degree of cardiac rotation exists. Such a condition results in a position of the heart that is more transverse than usual and may give rise to electrocardiograms suggestive of slight left ventricular preponderance. This status would be likely to occur in a broad-chested person or in one with a prominent abdomen. In a similar manner, a low diaphragm with little, if any, cardiac rotation and with the heart tending to assume a vertical position, might result in electrocardiograms representing slight right ventricular preponderance. This would be likely to occur in a long, narrow-chested person.

Changes which occur in the electrocardiogram from postural effects have been recorded. These alterations consist principally in an increased amplitude of deflection of the S wave in lead I, such as that which is observed in shifting the patient from the left to the right side.

In interpreting the curves of ventricular preponderance care must be taken not to confuse an exaggerated Q wave with the S wave. This appears to be a gross improbability; yet I have repeatedly seen this error made. For instance, the electrocardiogram recorded in Figure 6 displays considerable downward deflection of the QRS complex in lead III; yet the exaggerated downward deflection is that of the Q wave, not of the S wave, and the record is one of slight preponderance of the right ventricle, as shown by the exaggeration of the S wave in lead I.

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Chapter III

SINUS ARHYTHMIA

The simplest disturbance in cardiac rhythm is known as sinus, or respiratory, arrhythmia. In most instances it is a physiologic phenomenon, caused by vagal stimulation which occurs during the respiratory cycle and which influences the sino-auricular node. This results in transient slowing of the whole heart. A slight acceleration of rate occurs during inspiration and a slight retardation during expiration. The individual waves retain their normal characteristics and maintain proper relationship one to the other; the arrhythmia is characterized by the variations in the lengths of the cycles which are caused by the alternate acceleration and slowing of the rate. Electrocardiograms of sinus arrhythmia are illustrated in Figures 16 to 20.

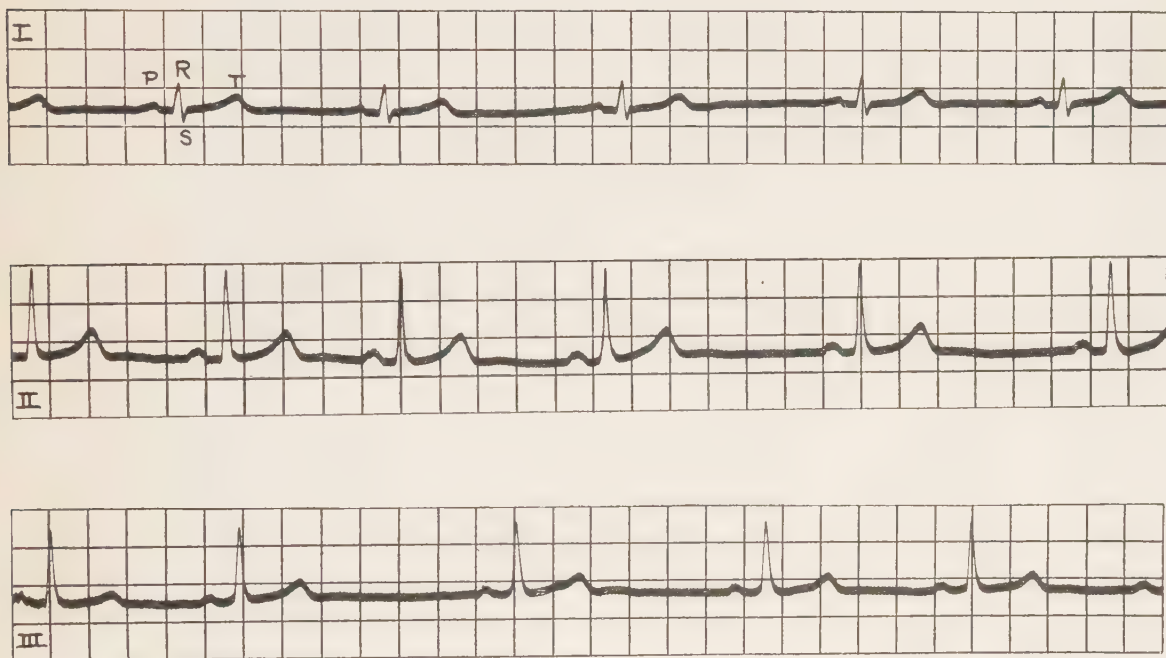


FIG. 16.—Record showing sinus arrhythmia. No change occurs in the character of the waves, or in their relationship to each other, but a phasic variation in the intervals between the R waves does occur. Record obtained from a man, aged twenty-six years, without evidence of heart disease.

The occurrence of this phenomenon in perfectly normal persons is common. It is especially likely to appear in children and young adults, although it may occur at any age. Sometimes it occurs temporarily following physical exertion.

Sinus arrhythmia is also observed when the heart is diseased but the occurrence then is probably only casual. In older persons it is likely to be associated with definite slowing in cardiac rate.

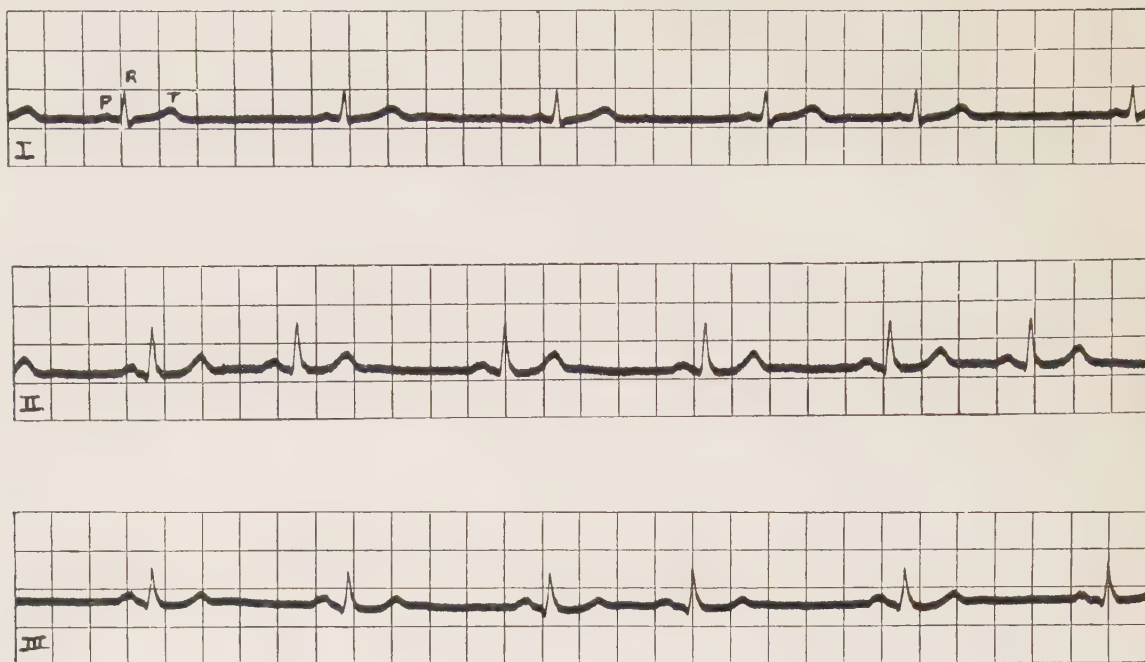


FIG. 17.—Record showing sinus arrhythmia. The complexes are normal, but a variation in the lengths of the cycles occurs. Record obtained from a man, aged sixty-four years, without evidence of heart disease.

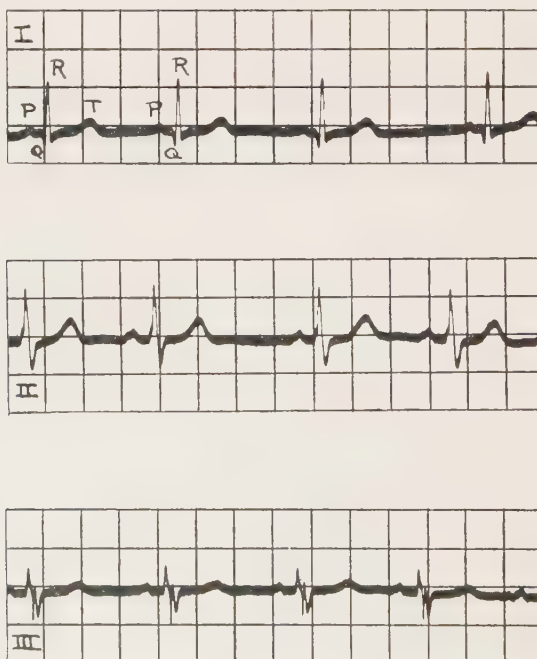


FIG. 18.—Record showing sinus arrhythmia. The waves all bear a normal relationship to one another, but a difference exists in the R-R intervals, which have a phasic tendency. Note the peculiar notching of the QRS complexes in lead III which exhibit phasic variations in amplitude. Record obtained from a boy, aged nine years, without evidence of heart disease.

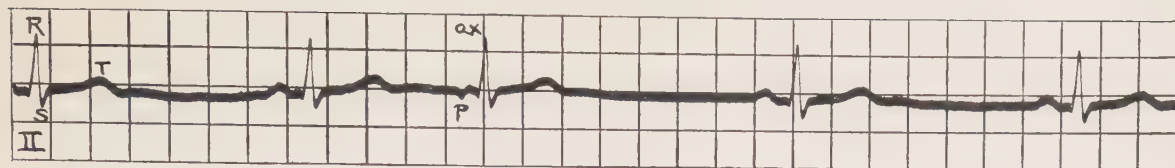


FIG. 19.—Record in lead II showing sinus arrhythmia interrupted by a premature auricular contraction. Note the inversion of the P wave in the complex which represents the premature contraction. Record obtained from a man, aged sixty years, with coronary sclerosis.

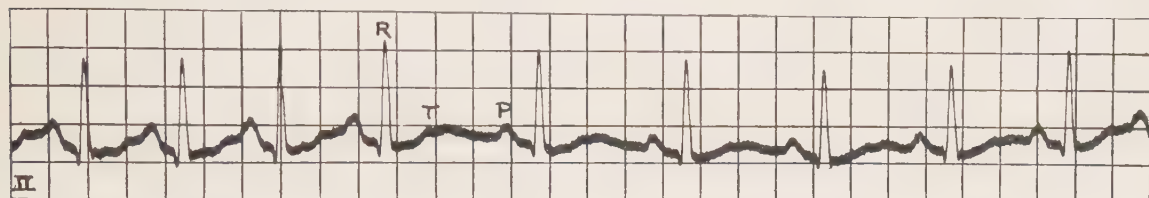


FIG. 20.—Record in lead II showing sinus arrhythmia. Note the variations in lengths of cycles. Record obtained from a man, aged thirty-two years, without evidence of organic heart disease.

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Chapter IV

PREMATURE CONTRACTIONS

Premature contractions, or extrasystoles as they are sometimes called, are one of the most common causes of cardiac arrhythmia during adult life. The exposed animal has been employed in numerous ways in the study of premature contractions; the usual method of investigation has been the application of induction shocks to various portions of the cardiac musculature. The induction shock applied during the diastolic period of the cardiac cycle produces a contraction which prematurely interrupts the cardiac rhythm. Stim-

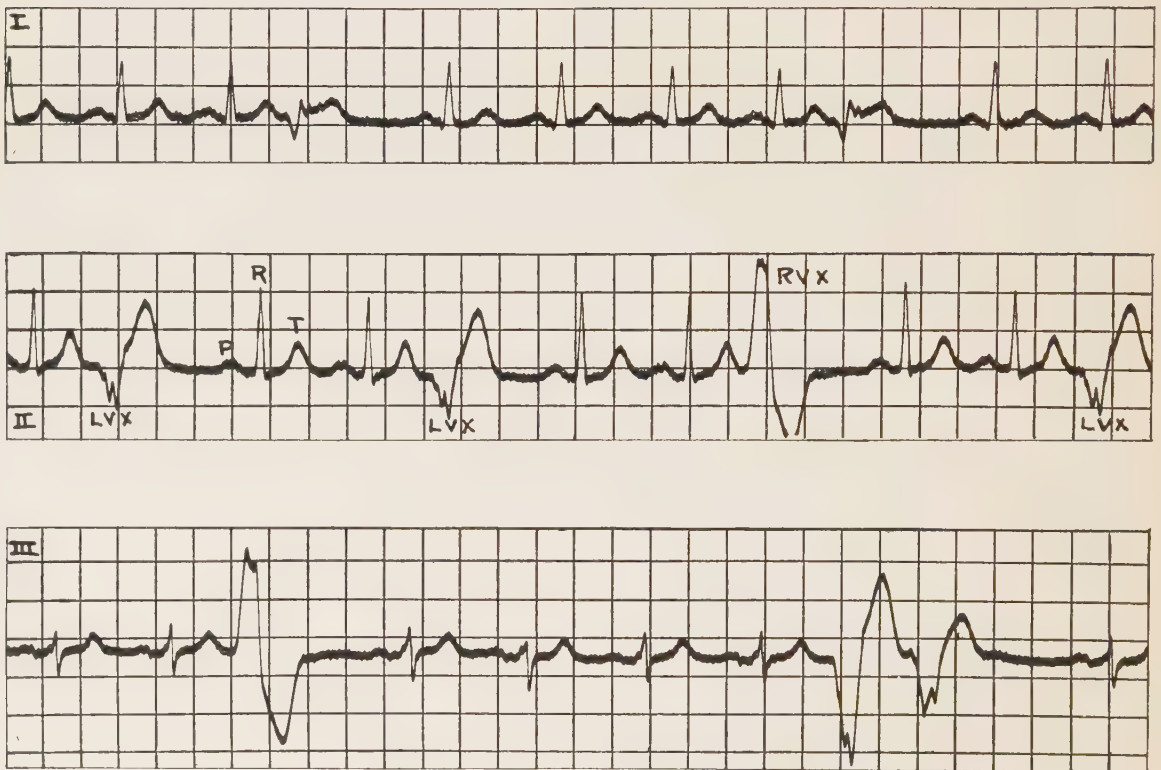


FIG. 21.—Record showing frequent right and left premature ventricular contractions. In lead III two premature contractions, which differ materially in form, occur in sequence. The record shows slight preponderance of the left ventricle. Record obtained from a man, aged forty-seven years, with exophthalmic goiter and essential hypertension.

ulation applied during the period of systole or contraction of a chamber is ineffectual, as the musculature is inexcitable or refractory at that time. The response of the heart to stimuli is not proportionate, but is always maximal, conforming to the “all or none” law of Bowditch. The beat which occurs prematurely is always followed by a pause, variable in length, which endures until the occurrence of the next beat of the normal cardiac rhythm. This period is known as the compensatory pause.

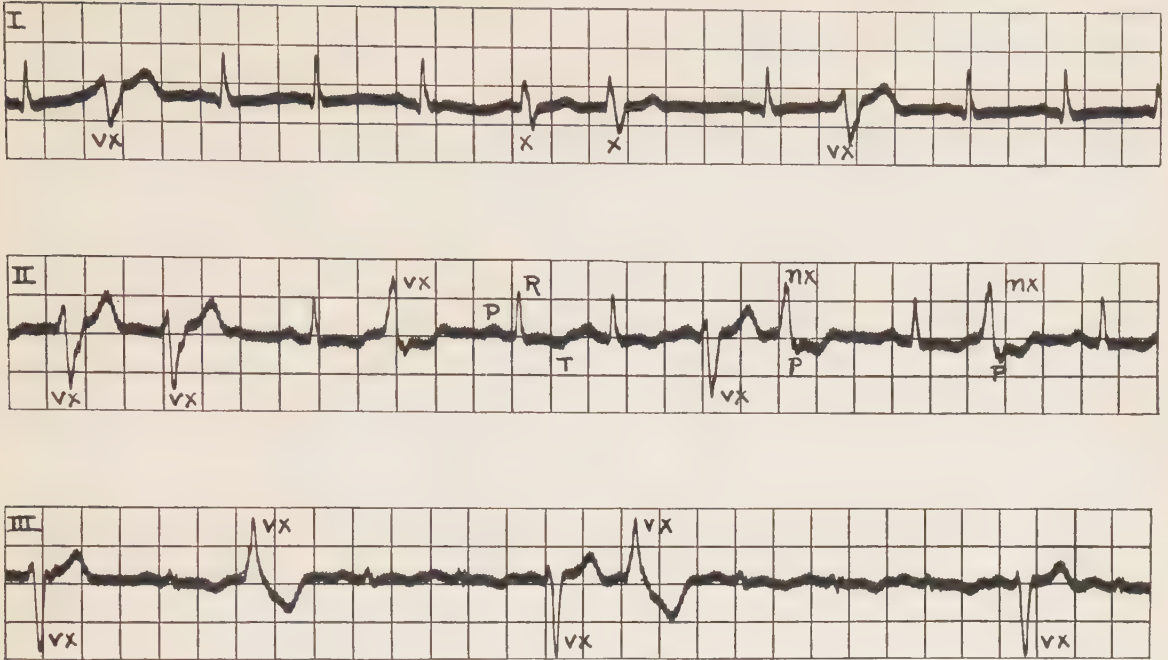


FIG. 22.—Record showing right and left premature ventricular contractions which arise from multiple foci of irritability. The differences in contour of similar premature contractions are apparent. Two premature nodal (A-V) contractions occur in lead II. The complexes of the dominant rhythm in lead III are of very low amplitude. The T waves in leads II and III are inverted. Record obtained from a woman, aged thirty-six years, with an irritable heart.

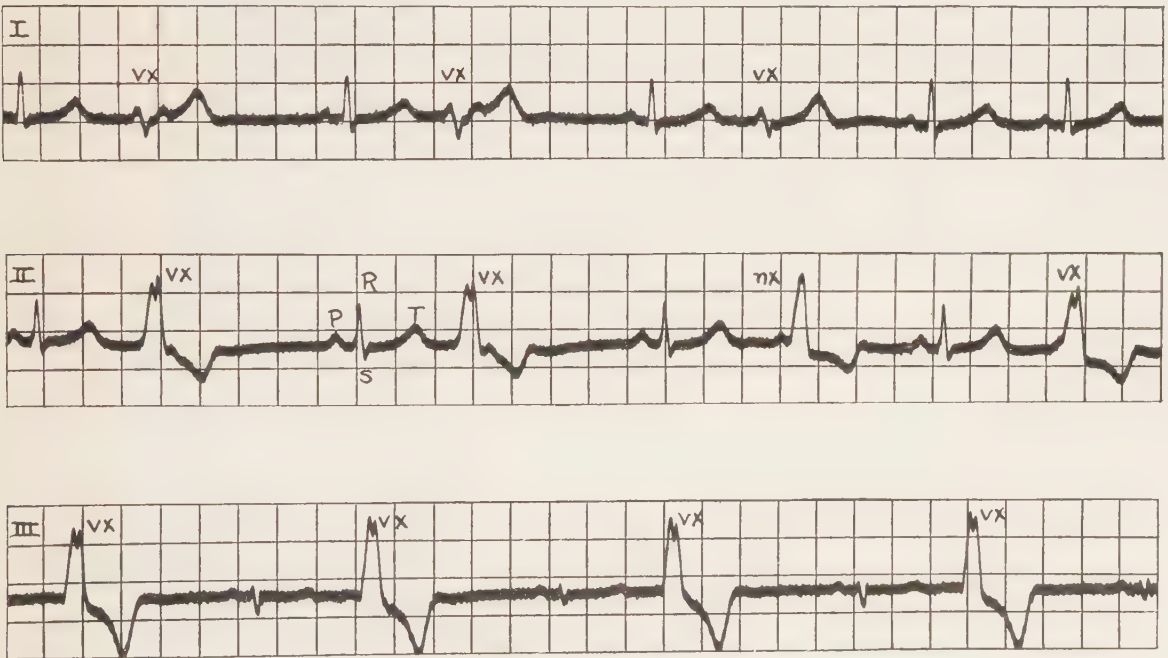


FIG. 23.—Record showing alternating premature contractions which arise in the right ventricle. The third premature contraction, which occurs in lead II, apparently arises in the junctional tissues; it has a definite P wave, but the P-R interval is greatly reduced, 0.07 second. Record obtained from a woman, aged forty-seven years, without demonstrable evidence of organic heart disease.

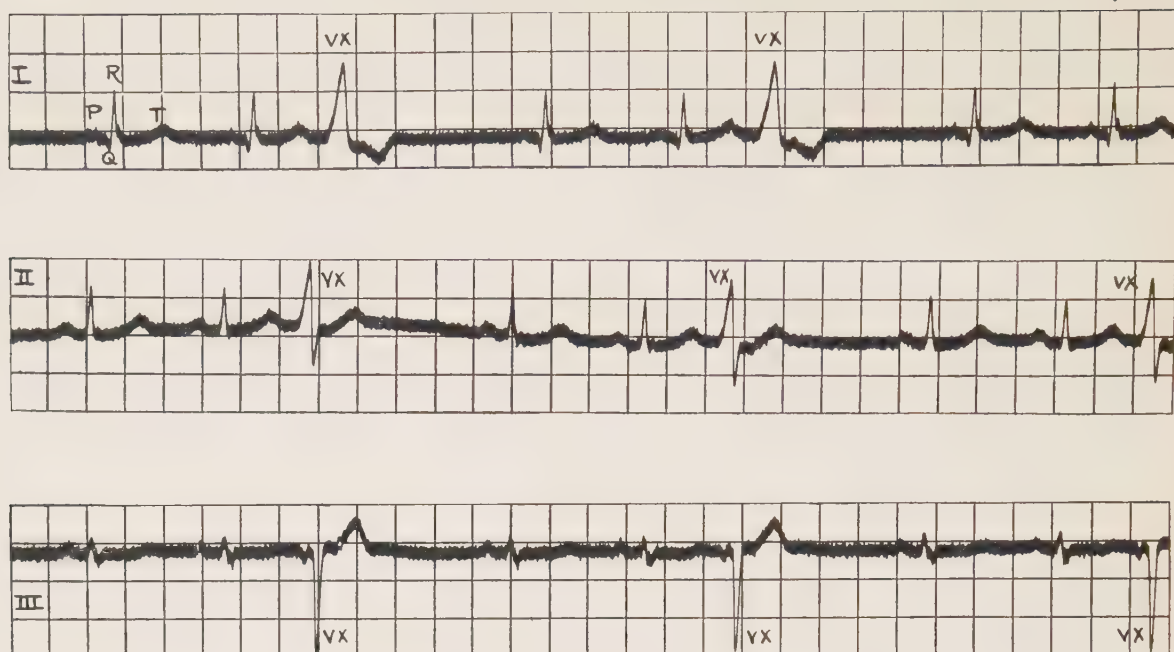


FIG. 24.—Record showing premature ventricular contractions which occur regularly every third beat. Record obtained from a man, aged sixty-three years, with mild essential hypertension.

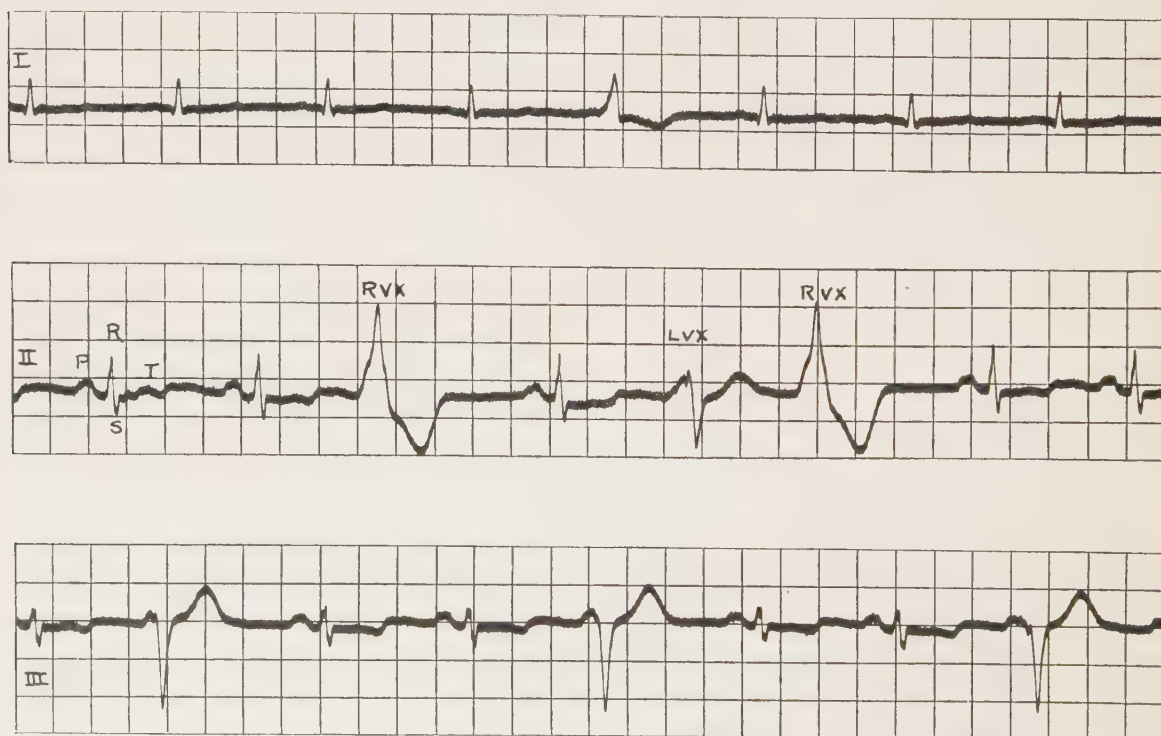


FIG. 25.—Record showing right and left premature ventricular contractions. In lead II both types are evident. Those which arise in the right ventricle are of high amplitude, with the T component directed downward, while the one which arises in the left ventricle is of lower amplitude and is directed upward; its T component is directed upward. The left premature ventricular contraction is immediately followed by one which arises in the right ventricle. The T waves of the dominant rhythm are inverted in leads II and III. Record obtained from a man, aged forty years, without evidence of organic heart disease.

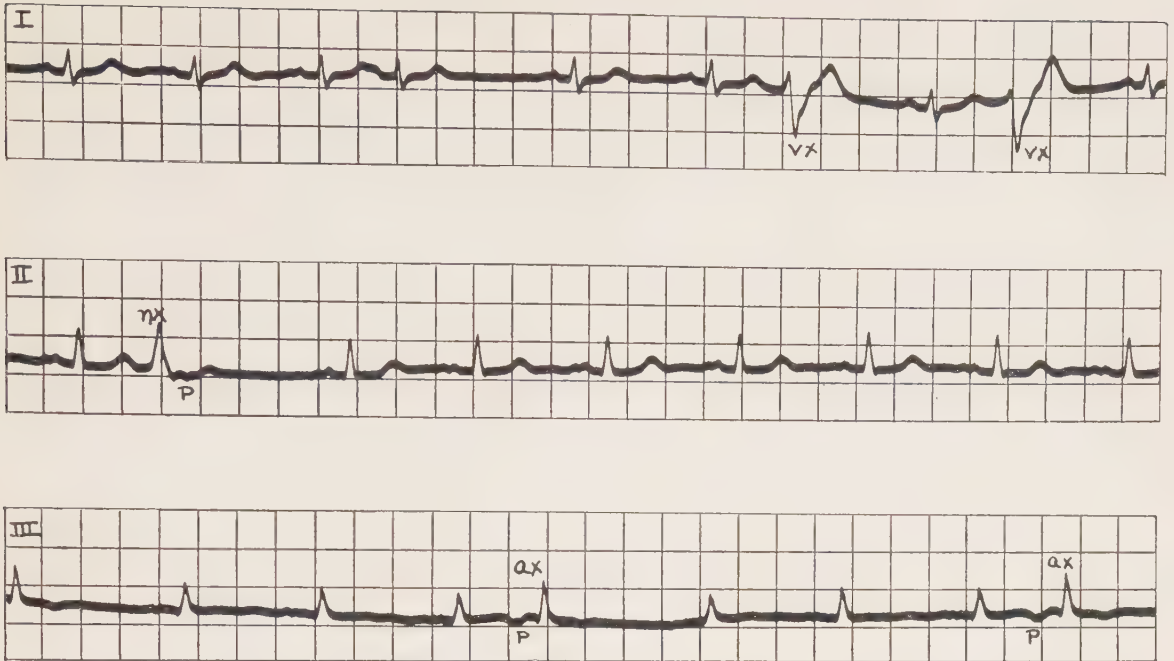


FIG. 26.—Record showing auricular, nodal (A-V), and premature ventricular contractions. The premature contractions which occur in lead III are auricular in origin; note the P wave inversions and the supraventricular form of ventricular complexes. The premature nodal (A-V) contraction occurs as the second complex in lead II. The P wave is inverted and follows the QRS complex, producing a notch in the accompanying T wave. Premature ventricular contractions, with bizarre complexes, are in evidence in lead I. Record obtained from a woman, aged fifty-seven years, with cardiac neurosis.

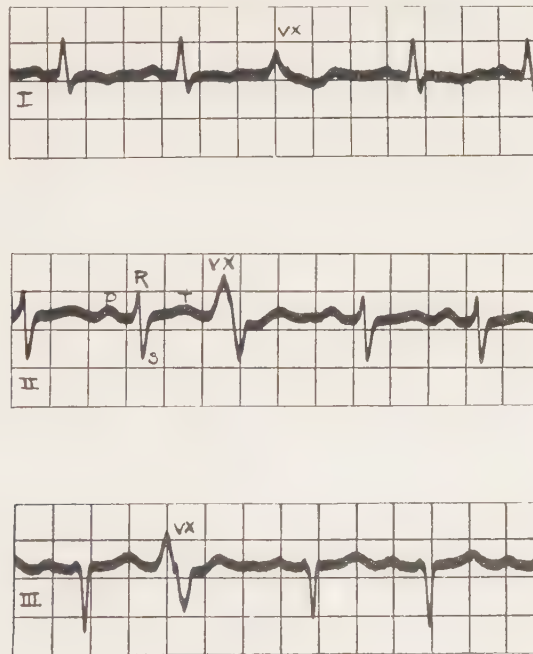


FIG. 27.—Record showing premature ventricular contractions which arise in the left ventricle and occur irregularly. The contour of these complexes is bizarre; and in lead II it is of increased amplitude, and is definitely diphasic, with the T component upright. The T waves in lead I are inverted and preponderance of the left ventricle is evident. Record obtained from a man, aged sixty-seven years, with hypertensive and coronary heart disease.

Premature contractions may arise in any portion of the ventricles, auricles or auriculo-ventricular junctional tissues. They are ectopic contractions which arise from foci or

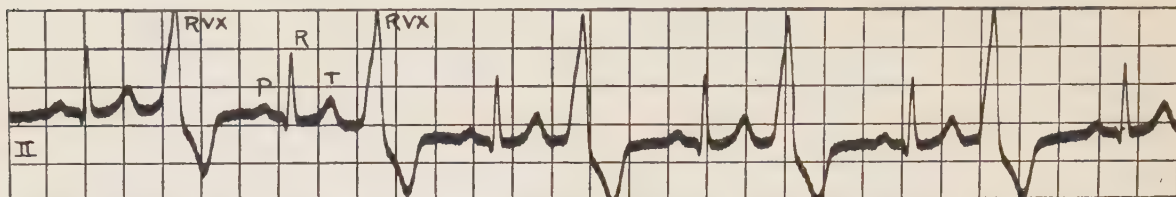


FIG. 28.—Record in lead II showing alternate right premature ventricular contractions. Note the regularity of the interruptions. Record obtained from a man, aged fifty-eight years, with hyperfunctioning adenomatous goiter.

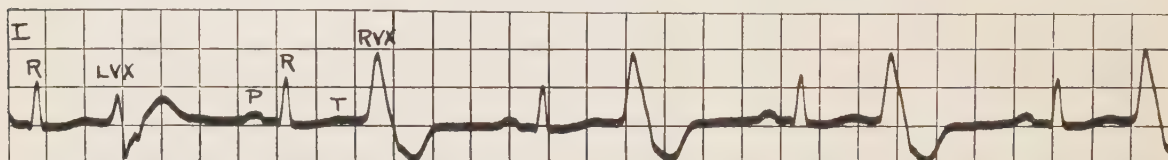


FIG. 29.—Record in lead I showing right and left premature ventricular contractions. Record obtained from a woman, aged fifty-two years, with hypertensive heart disease.

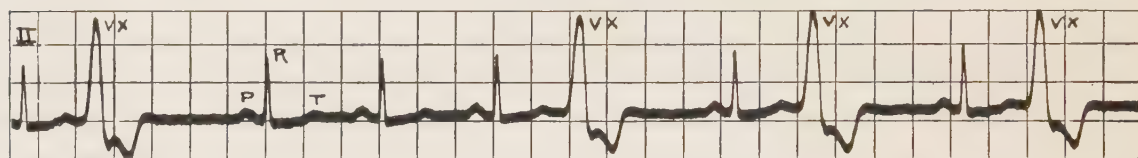


FIG. 30.—Record in lead II showing premature ventricular contractions which arise in the right ventricle. The amplitude of the complexes is markedly increased and directed upward, whereas the T components are opposite in direction. In the latter half of the record the premature contractions alternate with normal complexes. Note the prematurity of the beats and the well-marked compensatory pauses. Record obtained from a man, aged forty-three years, with cardiac neurosis.

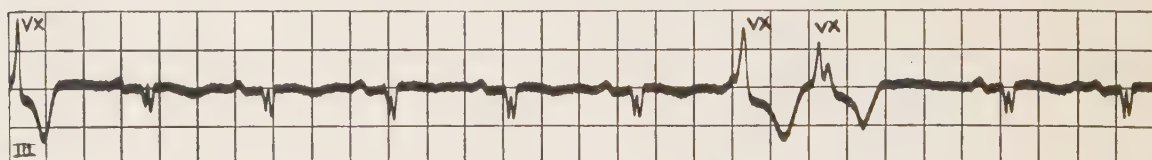


FIG. 31.—Record in lead III showing isolated and successive premature ventricular contractions. Note the "W" notching of the QRS complexes of the dominant rhythm. Record obtained from a woman, aged sixty-two years, with diabetes mellitus of the vascular type.

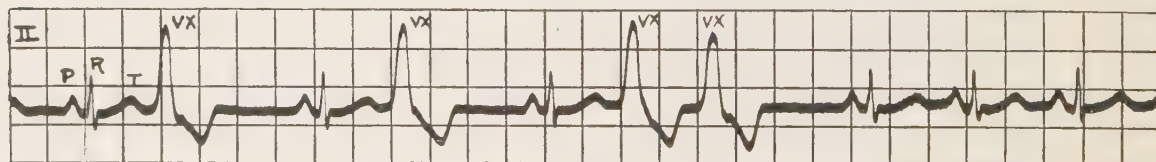


FIG. 32.—Record in lead II showing irregularly placed premature contractions arising in the right ventricle. Note the sequence of two premature contractions which vary slightly in form. Record obtained from a woman, aged forty-four years, without evidence of organic heart disease.

stimuli in areas other than the sino-auricular node. They may appear regularly, interrupting the rhythm at definitely recurrent intervals, or their appearance may be hap-

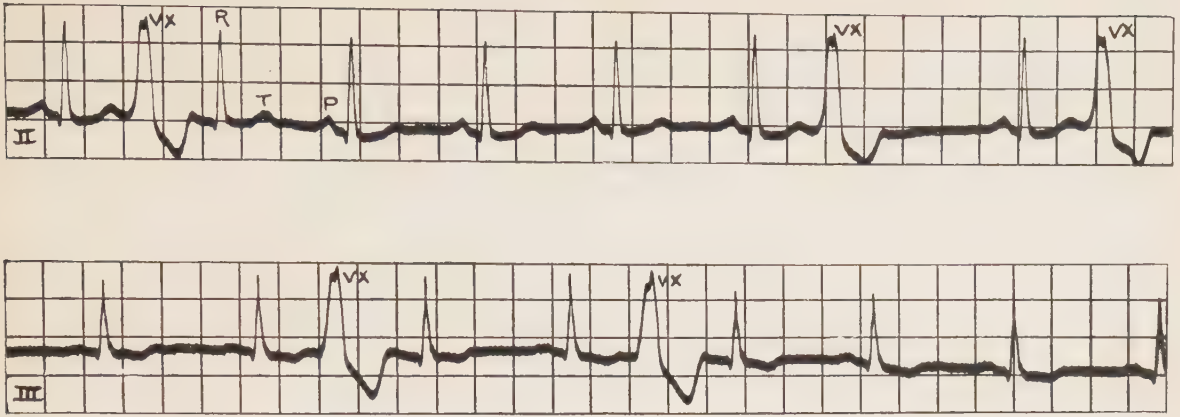


FIG. 33.—Record in leads II and III showing irregularly placed, right premature ventricular contractions. Note the uniformity of character of the premature contractions in similar leads and the interpolated contractions in lead III. Record obtained from a man, aged forty-one years, without evidence of organic heart disease.

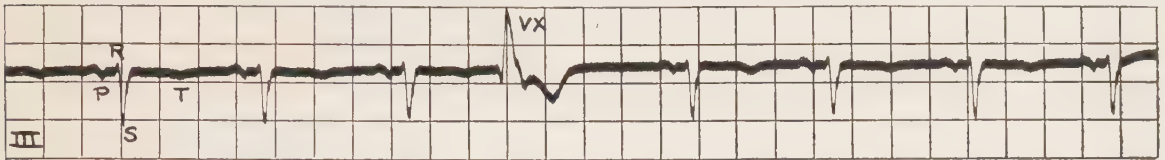


FIG. 34.—Record in lead III showing a premature ventricular contraction. Note the inversion of the T and P waves. Record obtained from a man, aged sixty-two years, with coronary sclerosis accompanied by angina pectoris.

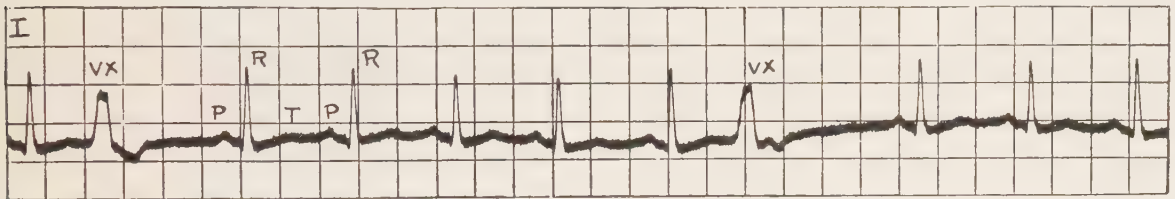


FIG. 35.—Record in lead I showing premature ventricular contractions. Slight differences in contour of the complexes occur, and the second premature contraction is followed by a longer compensatory pause. Record obtained from a woman, aged forty years, with secondary anemia.

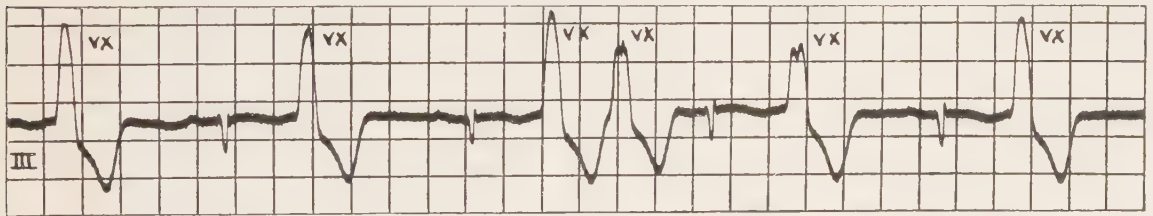


FIG. 36.—Record in lead III showing premature ventricular contractions which arise from two distinct foci in the right ventricle. The difference in contour between the two forms of premature contractions is apparent. Note the occurrence of two consecutive premature contractions from different areas. The general trend of the record is that of alternating premature contractions. Record obtained from a woman, aged thirty-five years, with cardiac neurosis.

hazard. Their occurrence every second beat is referred to as “coupled beats” or “pulsus bigeminus” (Figs. 23, 28, 29, and 49), and every third beat, as “pulsus trigeminus” (Figs. 24 and 25). They may occur regularly, at greater intervals, such as every fourth, fifth,



FIG. 37.—Record in lead III showing ventricular and nodal (A-V) premature contractions. Record obtained from a man, aged seventy-eight years, with essential hypertension.

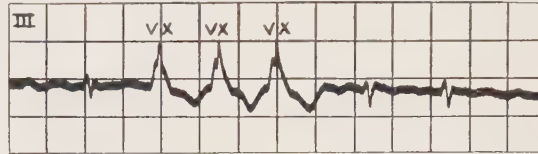


FIG. 38.—Record in lead III showing three successive premature ventricular contractions which interrupt the arrhythmia of auricular fibrillation. Record obtained from a woman, aged sixty-eight years, with hypertensive heart disease.

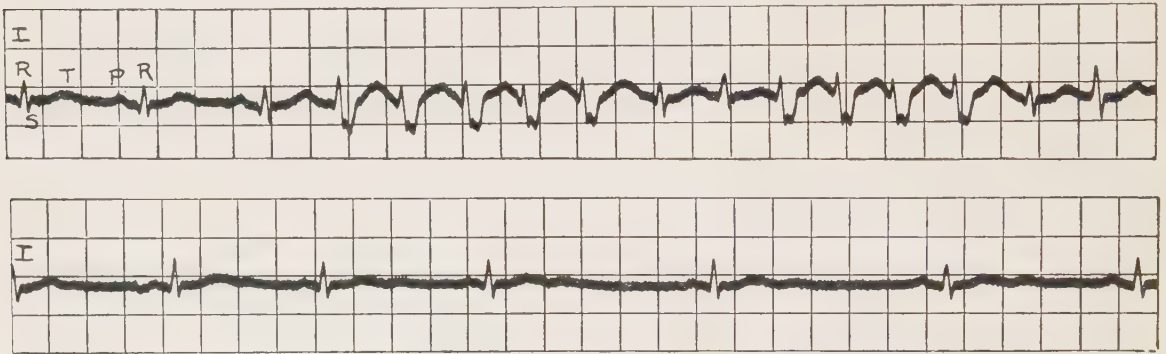


FIG. 39.—Continuous record taken in lead I. Short sequences of premature ventricular contractions are seen; with the restoration of sinus control a definite sinus arrhythmia is becoming established. Record obtained from a man, aged forty years, with slight cardiac hypertrophy of uncertain etiology.

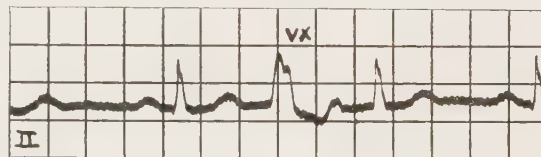


FIG. 40.—Record in lead II showing an interpolated beat which arises in the right ventricle. Note the equal distances between the peak of the abnormal complex and the preceding and succeeding peaks respectively. Record obtained from a man, aged forty-two years, without evidence of heart disease.

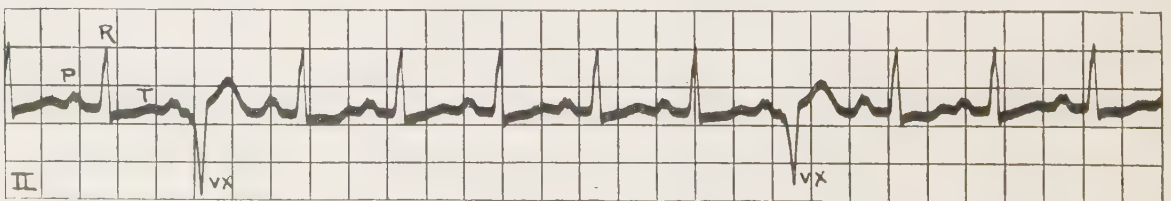


FIG. 41. Record in lead II showing two interpolated ventricular beats; note their occurrence at the time when the normal beat should occur. Record obtained from a man, aged twenty-seven years, with essential hypertension.

or sixth beat (Figs. 45 and 51). Most often their occurrence is so lacking in uniformity that they give rise to a rhythm that is almost totally irregular (Figs. 21, 22, 25, 30, 32,

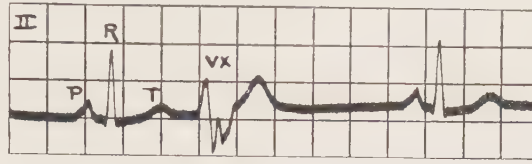


FIG. 42.—Record in lead II showing a right premature ventricular contraction which has considerable deformity of contour. Record obtained from a man, aged fifty years, without evidence of heart disease.

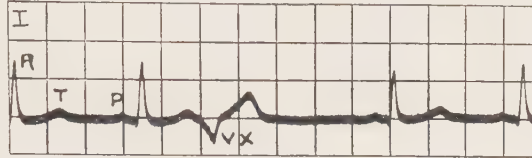


FIG. 43.—Record in lead I showing a premature ventricular contraction of peculiar form. Record obtained from a man, aged sixty-three years, with adenomatous goiter.

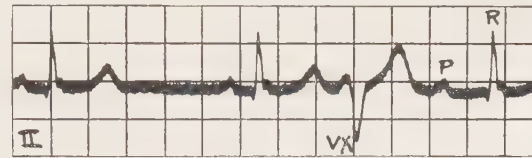


FIG. 44.—Record in lead II showing a premature ventricular contraction with temporary delay in auriculoventricular conduction during the phase of recovery. Record obtained from a man, aged forty-four years, with cardiac neurosis.

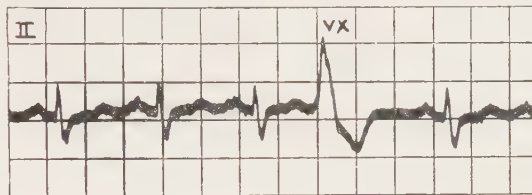


FIG. 45.—Record in lead II showing one right premature ventricular contraction; note its prematurity and its compensatory pause. The premature contraction has a rather tall, deformed, upright QRS complex and an angular T component directed downward. Record obtained from a man, aged seventy years, with hypertensive and coronary heart disease.

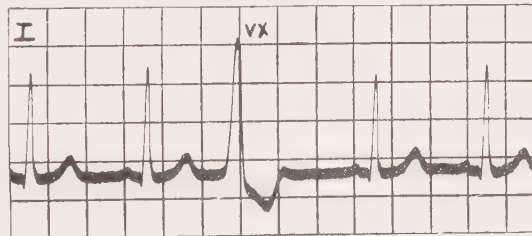


FIG. 46.—Record in lead I showing one premature ventricular contraction of high amplitude and with fairly normal contour. Record obtained from a woman, aged fifty-eight years, with moderate essential hypertension.

33, 36, and 37). At times they occur as only occasional and transient interruptions of the regular rhythm (Figs. 26, 27, 34, 35, 40, 41, 42, 43, 44, 45, 46, 47, 48, 51, 54, 55, 56, 57, 58, 59, 60, and 61).

The premature contractions may be paired, they may occur in threes or they may cause short periods of paroxysmal tachycardia (Figs. 31, 32, 38, 39, 52, and 53). At

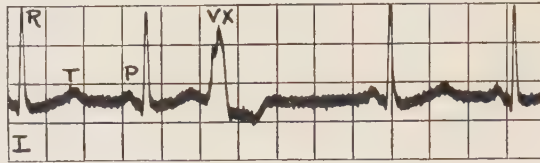


FIG. 47.—Record in lead I showing a premature ventricular contraction. There is marked aberration in form, but the QRS amplitude is less than that of the normal complexes. Record obtained from a man, aged fifty-nine years, with mild essential hypertension.

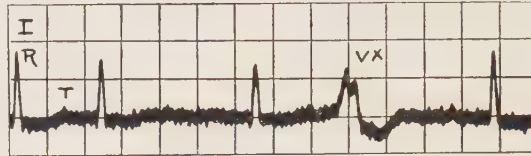


FIG. 48.—Record in lead I showing a premature ventricular contraction of low amplitude. The aberrant complex has a definitely widened base, a notched apex, and the T component is inverted. Considerable body tremor is evident in this record which was obtained from a man, aged seventy-four years, with coronary sclerosis.

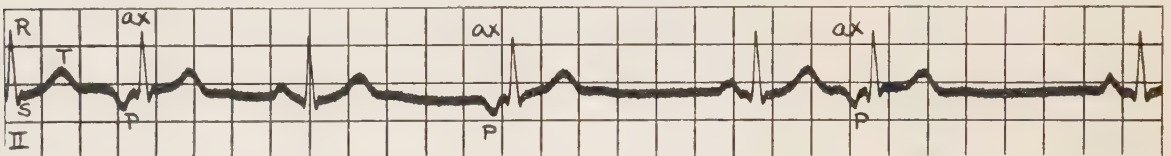


FIG. 49.—Record in lead II showing premature auricular contractions. The P waves in the abnormal complexes are inverted, whereas the ventricular response results in QRS complexes of normal appearance. Record obtained from a man, aged thirty-six years, with cardiac neurosis.

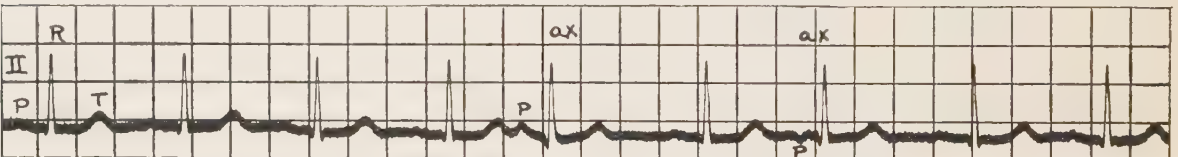


FIG. 50.—Record in lead II showing premature auricular contractions. The first premature contraction, which occurs as the fifth complex in the record, differs from the normal complexes only in its prematurity. The P wave of this complex is upright, indicating that its point of origin is at, or near, the sino-auricular node. The second premature contraction has an inverted P wave; this is evidence of a focus in the auricle away from the sino-auricular node. Record obtained from a man, aged seventy-two years, with coronary sclerosis.

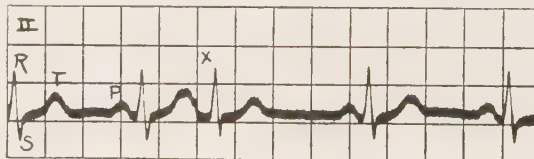


FIG. 51.—Record in lead II showing a premature contraction apparently arising near the sino-auricular node. Record obtained from a man, aged sixty-four years, with coronary sclerosis.

times, particularly when the cardiac rate is slow, a ventricular beat may occur before the succeeding stimulus arrives from the auricles and may fall quite evenly between two normal beats; it is then said to be interpolated (Figs. 33 and 41).

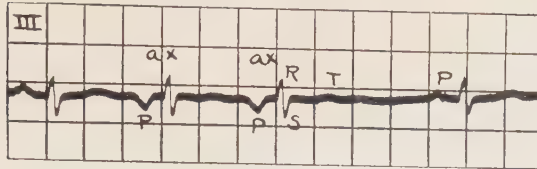


FIG. 52.—Record in lead III showing paired premature auricular contractions. Note the inversion of the P waves of the premature complexes. Record obtained from a woman, aged forty-two years, with cardiac neurosis.

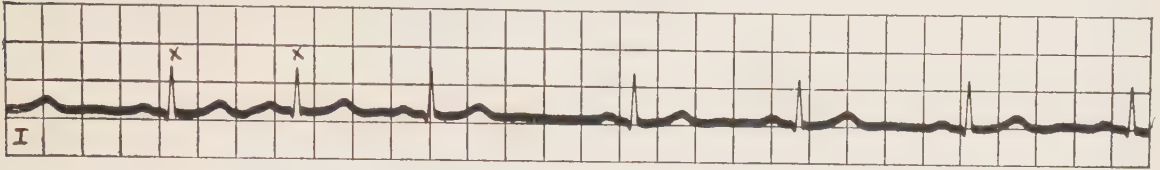


FIG. 53.—Record in lead I showing paired premature contractions arising at, or near, the sino-auricular node. The complexes are normal except for their prematurity. Record obtained from a woman, aged fifty-eight years, with essential hypertension.

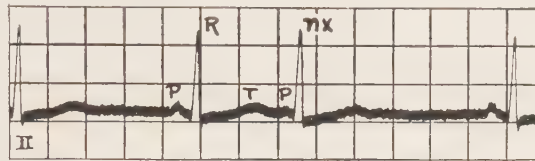


FIG. 54.—Record in lead II showing a premature nodal (A-V) contraction. Note the diminished P-R interval (0.05 second) of the premature contraction. The ventricular complex is normal. Record obtained from a woman, aged fifty-seven years, with hypertensive heart disease.

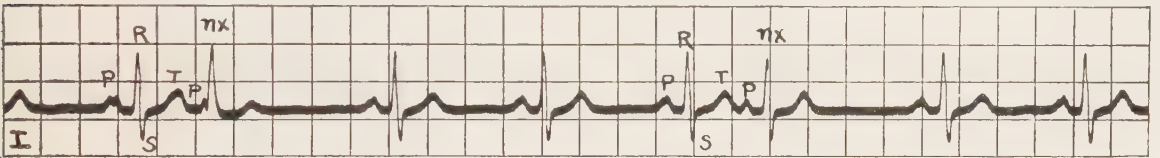


FIG. 55.—Record in lead I showing premature nodal (A-V) contractions with diminution in the P-R intervals. Note the extreme shortening of conduction time in the first premature contraction. Record obtained from a man, aged forty-six years, with essential hypertension.

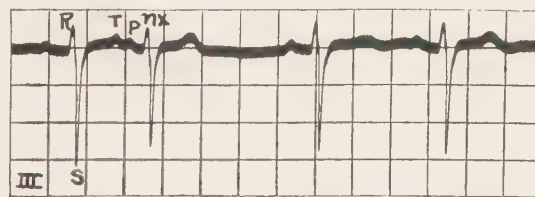


FIG. 56.—Record in lead III showing a premature nodal (A-V) contraction with diminished P-R interval. Record obtained from a man, aged seventy-five years, with coronary and hypertensive heart disease.

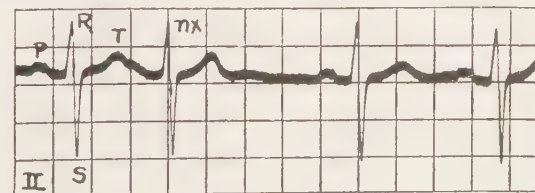


FIG. 57.—Record in lead II showing a premature nodal (A-V) contraction. Note the absence of the P wave, the auricle and ventricle contracting synchronously. Record obtained from a man, aged forty-seven years, with coronary sclerosis accompanied by angina pectoris.

It is not unusual to see represented in the same record premature contractions which have arisen in each of several foci of increased irritability (Figs. 22, 23, 26, and 37).

The presence of premature contractions does not indicate organic heart disease. Premature contractions are frequently observed in perfectly healthy individuals and are undoubtedly the expression of increased cardiac irritability which may result from many causes. Their frequency in patients with frank cardiac neurosis establishes a defi-

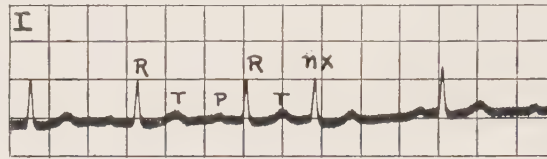


FIG. 58.—Record in lead I showing a premature nodal (A-V) contraction. Note the absence of the P wave in the premature complex; the QRS component exhibits a normal contour. Record obtained from a woman, aged sixty-nine years, with coronary sclerosis accompanied by angina pectoris.

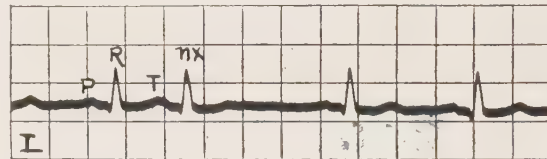


FIG. 59.—Record in lead I showing a premature nodal (A-V) contraction. The P wave is absent; apparently it falls synchronously with the R wave. Record obtained from a man, aged fifty-four years, with syphilitic aortitis and aortic insufficiency.

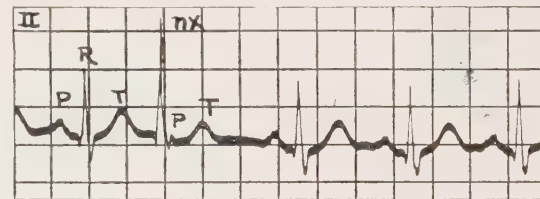


FIG. 60.—Record in lead II showing a premature nodal (A-V) contraction with evidence of retrograde conduction. Note that the P wave occurs just after the R wave. Record obtained from a man, aged fifty-one years, with exophthalmic goiter.

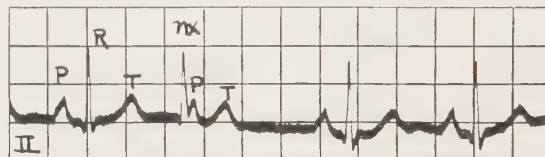


FIG. 61.—Record in lead II showing a premature nodal (A-V) contraction with retrograde conduction; the P wave falls just at the termination of the R wave and deforms its contour. Record obtained from a man, aged seventy-nine years, with coronary sclerosis.

nite relationship between this form of cardiac arrhythmia and other functional and anxiety states. The excessive use of tobacco, tea, coffee, and stimulant drugs has been noted as an exciting agent and withdrawal has resulted in the establishment of normal rhythm. Premature contractions may occur in patients with seriously affected hearts, but the incidence in such persons is not greater than in those whose hearts are not affected. I do not believe that it is ever justifiable to try to establish the diagnosis of heart disease from

the presence of premature contractions alone. The area of the heart which gives rise to these abnormal beats does not seem to influence their clinical significance.

PREMATURE VENTRICULAR CONTRACTIONS

Premature contractions may arise in any portion of the ventricular musculature, and, owing to the fact that such waves of excitation must traverse abnormal paths, the resulting electrocardiographic deflections are abnormal. The resulting deformity of the QRS complex and of the T wave is variable, depending apparently on the site in the ventricle where the abnormal stimulus was produced. Probably the degree of deformity of the complex depends on the remoteness from the customary avenues of conduction of the area of abnormal stimulation—the greater the remoteness, the greater the deformity. The QRS complex is frequently of very high amplitude and is diphasic in character with considerable deformity of contour; the deformity may consist in notching of the components of the complex and in an increase in its period of execution. The T component of the abnormal complex is frequently of increased amplitude, peaked, and at times directed downward (negative) (Fig. 34).

The generally accepted criteria for the differentiation of premature contractions which arise in the left and in the right ventricles are shown in Table 2. Those which arise

TABLE 2

THE GENERALLY ACCEPTED CRITERIA FOR THE DIFFERENTIATION OF LEFT AND RIGHT PREMATURE VENTRICULAR CONTRACTIONS

Lead.	Left ventricle.		Right ventricle.	
	QRS.	T.	QRS.	T.
I.	Upright.	Downward.	Downward.	Upright.
II.	Downward.	Upright.	Upright.	Downward.
III.	Downward.	Upright.	Upright.	Downward.

in the left ventricle usually are characterized by the abnormal QRS complex being directed upward and the T component, downward in lead I. The complexes are just reversed in leads II and III. Premature contractions which arise in the right ventricle display complexes in the respective leads just opposite in direction to those which arise in the left ventricle. Occasionally the direction of deflection in lead I may be reversed. Examples of left and right premature ventricular contractions are shown in Figures 21, 22, and 25. Premature contractions which arise in different portions of the same ventricle will differ in character; the differences may appear in the amplitude of deflection, the duration of the complexes and the degree of deformity (Figs. 26, 31, 32, 35, 36, and 37).

The type and degree of deformity of complex is extremely variable; the deflections often are of great amplitude, with notching of the ascending or descending limbs, or of the apex, and with greatly prolonged conduction time (Figs. 21, 23, 25, 28, 29, 30, 32, 33, 36, 37, and 45). At times the complexes are deformed but little and display only great amplitude of deflection with increased duration of conduction time (Figs. 22, 24, 41, 44, and 46). It is not unusual to observe premature ventricular contractions the amplitude of deflection of which is not increased; they may be even considerably less than the complexes of the

normal rhythm but they are definitely deformed in contour (Figs. 27, 35, 40, 42, 43, 44, 47, and 48).

The P wave of the abnormal complex is frequently absent, submerged in the aberration. At times, however, it is recognizable in its proper place.

PREMATURE AURICULAR CONTRACTIONS

The ectopic focus responsible for the production of premature auricular contractions may be in any portion of the auricular musculature. In most instances, the QRS complex of the premature beat is normal in all respects and is so owing to the fact that the ventricle has responded to a supraventricular impulse which, beyond its point of origin, has traversed normal paths of conduction. If the point of ectopic stimulation in the auricle is remote from the sino-auricular node, the P wave of the premature complex will be inverted (negative) (Figs. 26, 49, 50, and 52).

However, if the premature contraction arises from a focus of stimulation within the immediate vicinity of the sino-auricular node, the resulting complex may be entirely normal, with the P wave upright (positive) and with the proper relationship in time to the other components. A complex of this sort can be identified only by its prematurity (Figs. 50, 51, and 53). Occasionally premature auricular contractions may exhibit deformity of the accompanying ventricular complex. This has been shown to occur especially when the regular rhythm is interrupted extremely prematurely. Figure 51 shows the P wave of the premature complex occurring before the completion of the preceding T wave and definitely deforming its contour.

PREMATURE NODAL (A-V) CONTRACTIONS

Premature contractions which arise in the auriculoventricular bundle, or node, are referred to as "premature nodal contractions." Owing to the fact that the area of formation of the ectopic impulse occupies an intermediary position between auricles and ventricles, a considerably shorter path of conduction is necessitated for the impulse to provoke a ventricular response. Therefore, in many cases of premature nodal contractions, the auriculoventricular, or P-R interval, is greatly reduced, not infrequently to 0.04 second. The ventricular complex of the premature beat may be entirely unaltered, as the provoking impulse reaches it from the supraventricular tissues (Figs. 54, 55, 58, and 59). At other times the ventricular complex may show variations in amplitude and in conduction time (Figs. 23, 37, and 56). At other times, when the area of production of the abnormal stimulus is lower in the junctional tissues, auricles and ventricles may contract simultaneously. Here the P wave is apparently absent, but on careful scrutiny it will be found to be superimposed on the R wave, after it has given rise to a complex of slightly greater amplitude than that which occurs in the normal rhythm. The amplitude will be increased especially when the peaks exactly coincide (Figs. 26, 57, 58, and 59).

When the ectopic focus is very low in the junctional tissues, the ventricles may contract in advance of the auricles; in such instances the ventricles are activated by retrograde conduction. Here it is found that the P wave immediately follows the R wave and that occasionally the P wave occurs before the completion of the R wave and either deforms its descending limb or occurs just after its completion. Under these conditions,

the P wave is invariably inverted and the QRS complex is often bizarre in contour (Figs. 22, 60, 61, and 62).

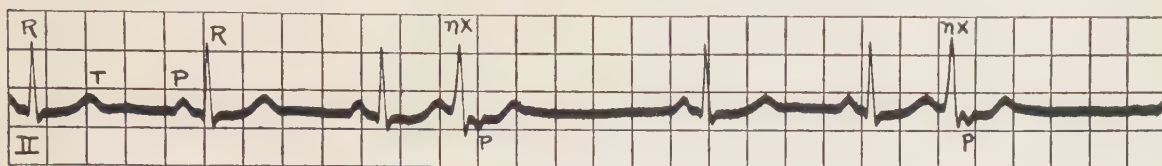


FIG. 62.—Record in lead II showing premature nodal (A-V) contractions. Both ventricle and auricle contract prematurely, but contraction of the ventricle precedes that of the auricle. The P wave, which is inverted, occurs after the QRS complex is completed and deforms the beginning of the T wave. Record obtained from a man, aged fifty-five years, without evidence of organic heart disease.

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Chapter V

AURICULAR FLUTTER

Auricular flutter is a not uncommon disturbance of cardiac activity. An acceleration of the auricles occurs which exceeds 200 a minute, and at times an auricular rate of 375 to 390 is assumed. In the majority of cases a varying degree of auriculoventricular block occurs; the ventricles may respond to every second, third, fourth, or fifth impulse, and occasionally complete auriculoventricular dissociation exists. Not infrequently the ventricular response is variable, owing to continually changing periods of block. This results in a totally irregular rhythm. Occasionally the ventricles respond to every auricular beat. The failure of auriculoventricular coördination is not attributable to disease of the junctional conducting system but, apparently, to the inability of the bundle to conduct all the impulses that are discharged so rapidly. When complete auriculoventricular dissociation occurs, independent of the action of digitalis, it may be assumed that disease of the conducting system exists.

There is apparently no difference pathologically between hearts which exhibit auricular rates under 200 (auricular tachycardia) and those with auricular rates over 200 (auricular flutter); yet, the clinical features of the cases and the electrocardiograms are so different as to justify the identification of auricular flutter as a definite entity.

Auricular flutter may occur as a fairly permanent disturbance of cardiac action or it may occur as a distinctly paroxysmal disorder, with an abrupt onset and a correspondingly abrupt termination.

The disturbed mechanism responsible for auricular flutter is extremely interesting, and it is important that its true nature be comprehended. Lewis and his coworkers solved this problem, and their conclusions, attained following exhaustive and careful experimental work, are undoubtedly correct. Following the rapid, rhythmic application of induction shocks, at varying rates, to the auricle of the exposed animal heart, electrocardiograms were obtained which were identical with those observed in clinical flutter. An unusual degree of regularity of auricular activity was observed, and the lengths of the cycles did not vary more than 0.001 to 0.003 second. It was found that the spread of the wave of excitation through the auricular mass was extremely regular and that the activation of some portion of the auricular tissue occurred during the whole cycle. It was further demonstrated that when the mechanism of flutter was once established following stimulation, its continuation was entirely independent of the original stimulus and that the flutter was caused by a wave continuously travelling through the auricular mass. The course taken by the wave was found to be around the opening of the superior vena cava, and at times around the openings of both the superior and the inferior venæ cavae, and that the direction was either upward or downward. The wave constituted a continuous circulating mechanism or circus movement. The wave of contraction, which had traversed a portion of the auricular wall, returned to its point of inception when the process of contraction had been entirely completed and was again capable of undergoing contraction. The repetition of these events constitutes circus movement.

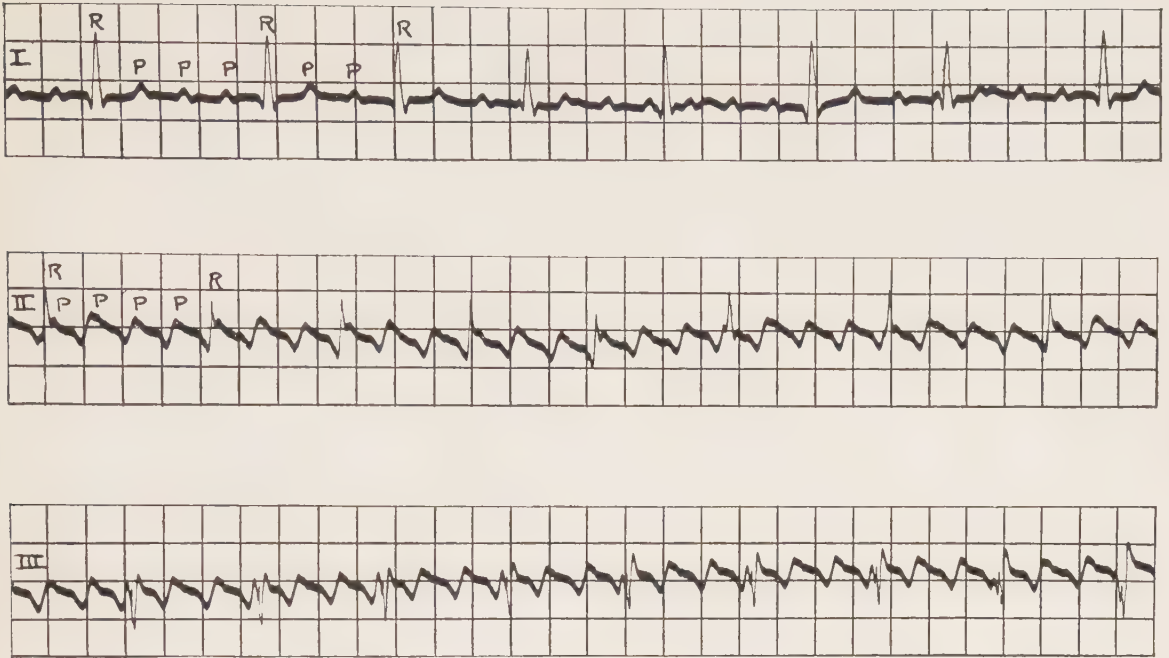


FIG. 65.—Record of auricular flutter with 3 : 1 and 4 : 1 block. The ventricular rate is 80, and the auricular rate is 270 contractions each minute. This record is somewhat unusual in its clear representation of the P waves in lead I. Record obtained from a man, aged forty-nine years, with exophthalmic goiter.

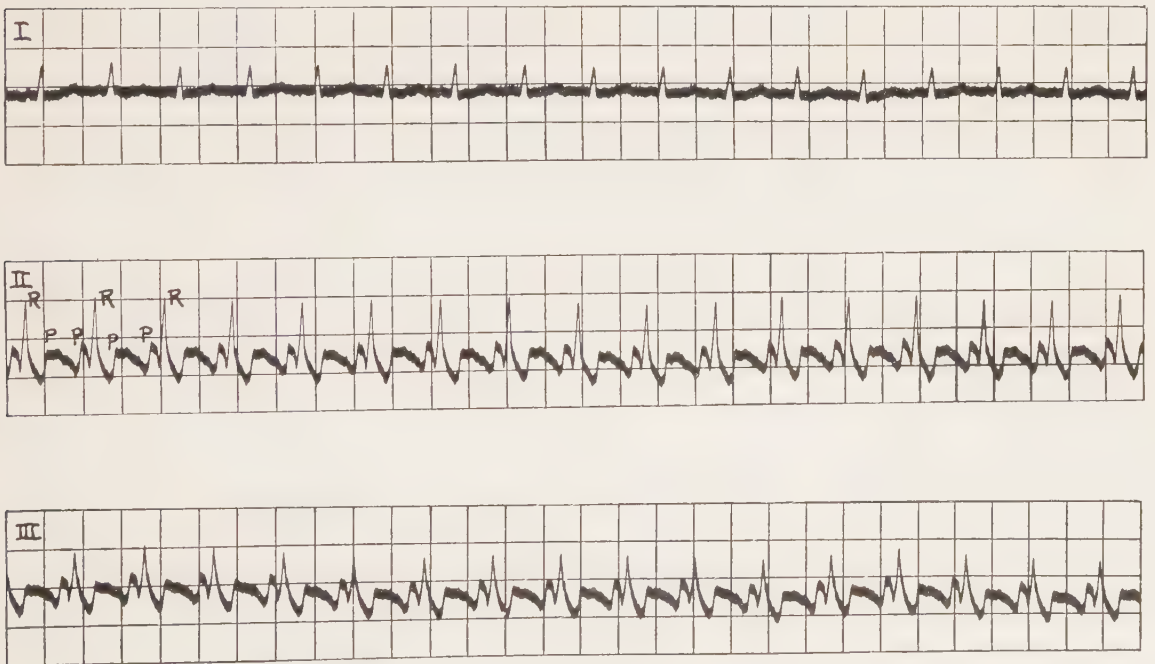


FIG. 66.—Record showing auricular flutter with 2 : 1 block. Note the great regularity of the P waves and their abrupt angulation. The auricular rate is 330, and the ventricular rate 165 contractions each minute. Record obtained from a man, aged sixty-six years, with exophthalmic goiter.

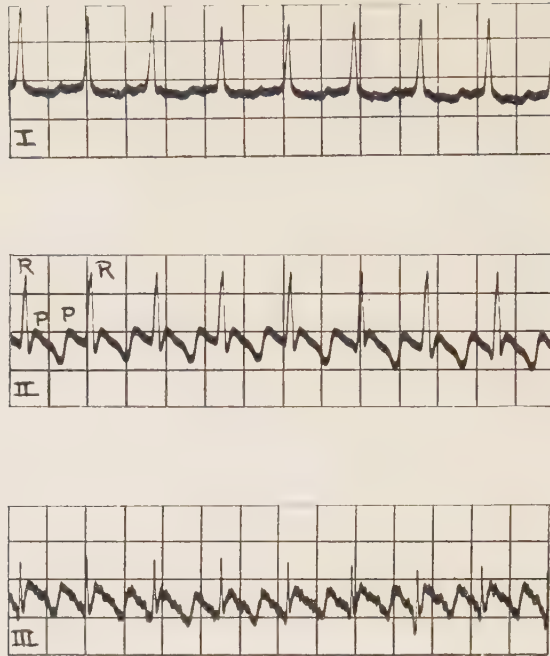


FIG. 67.—Record showing paroxysmal auricular flutter with 2 : 1 block. Note the angular P waves of high amplitude and of remarkable regularity which are observed particularly in leads II and III. Irregular increases occur in the depth of the Q deflections in lead III. The ventricular rate is 172, and the auricular rate is 344 contractions each minute. Record obtained from a woman, aged fifty-five years, with exophthalmic goiter.

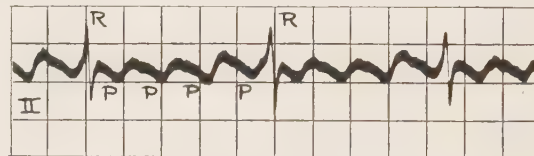


FIG. 68.—Record in lead II showing auricular flutter with 4 : 1 block. The ventricular rate is 63 and the auricular rate 252 contractions each minute. Note the regularity of the prominent P peaks. Record from a man, aged fifty-eight years, with hypertensive heart disease.

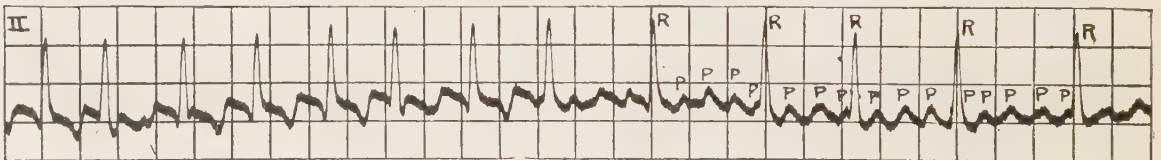


FIG. 69.—Record in lead II showing auricular flutter with 2 : 1, 3 : 1, 4 : 1, and 5 : 1 block. It is unusual to see such a variation in the degree of block in a brief period of time. Record obtained from a woman, aged forty-eight years, with exophthalmic goiter.

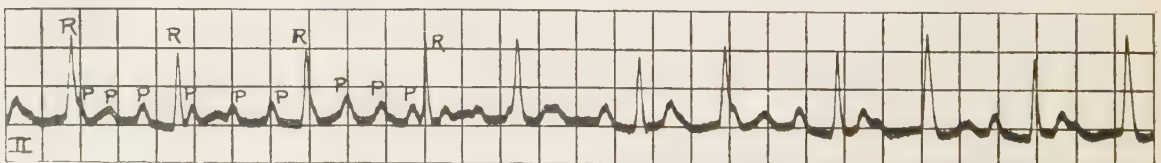


FIG. 70.—Record in lead II showing an unusual form of paroxysmal auricular flutter. Note the irregularity of the contour of the P waves and the varying degree of block. Record obtained from a man, aged forty years, with slight cardiac hypertrophy of indeterminate origin.

Lewis has called attention to variations in the regularity of the mechanism of flutter. He has denoted those cases which exhibit great regularity as "pure flutter" and those in

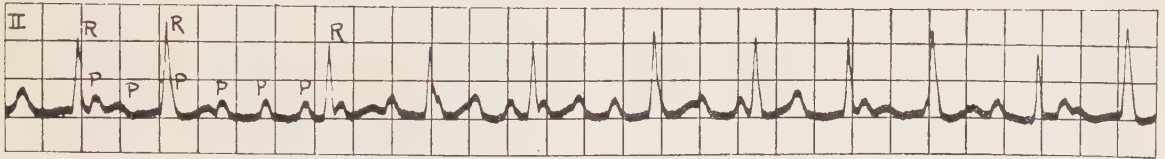
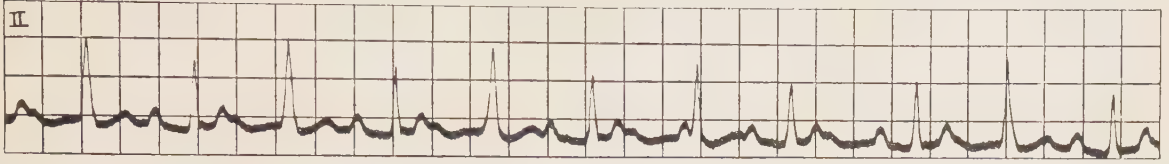


FIG. 71.—Records of the same patient who is represented in Figure 70, taken, in this instance, in lead II. The records show the peculiar form of flutter with varying degrees of block.

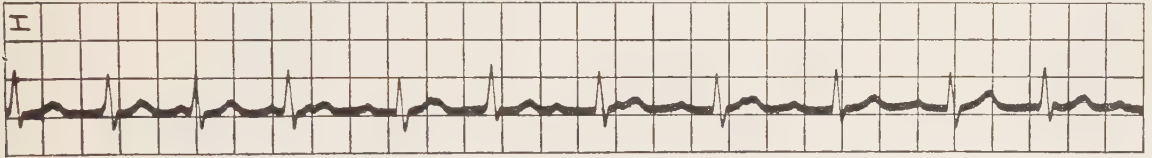


FIG. 72.—Record of the same patient who is represented in Figures 70 and 71, taken, in this instance, in lead I. As usual this lead shows less evidence of the condition.

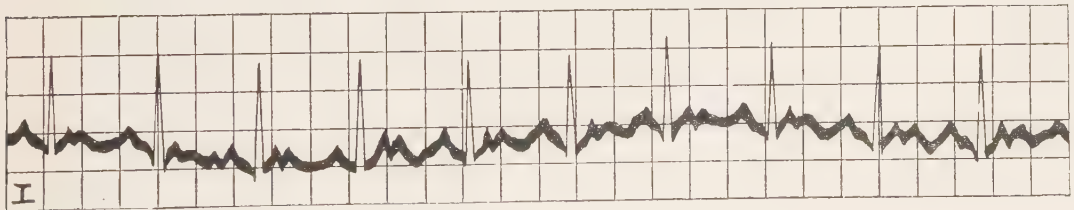
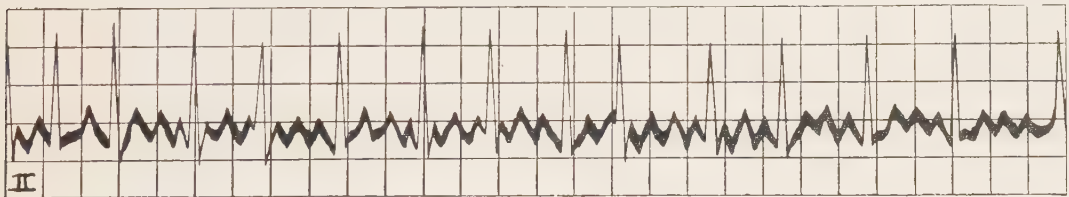


FIG 73 —Records in leads I and II showing auricular flutter with varying degrees of heart-block. Record obtained from a woman, aged forty-one years, with exophthalmic goiter.**

which variations occur from cycle to cycle, as "impure flutter." Impure flutter results when slight variations in the general path of the wave occur.

** Two asterisks follow the legend that appears beneath each of those records that were reproduced from old tracings printed on bronzed paper.

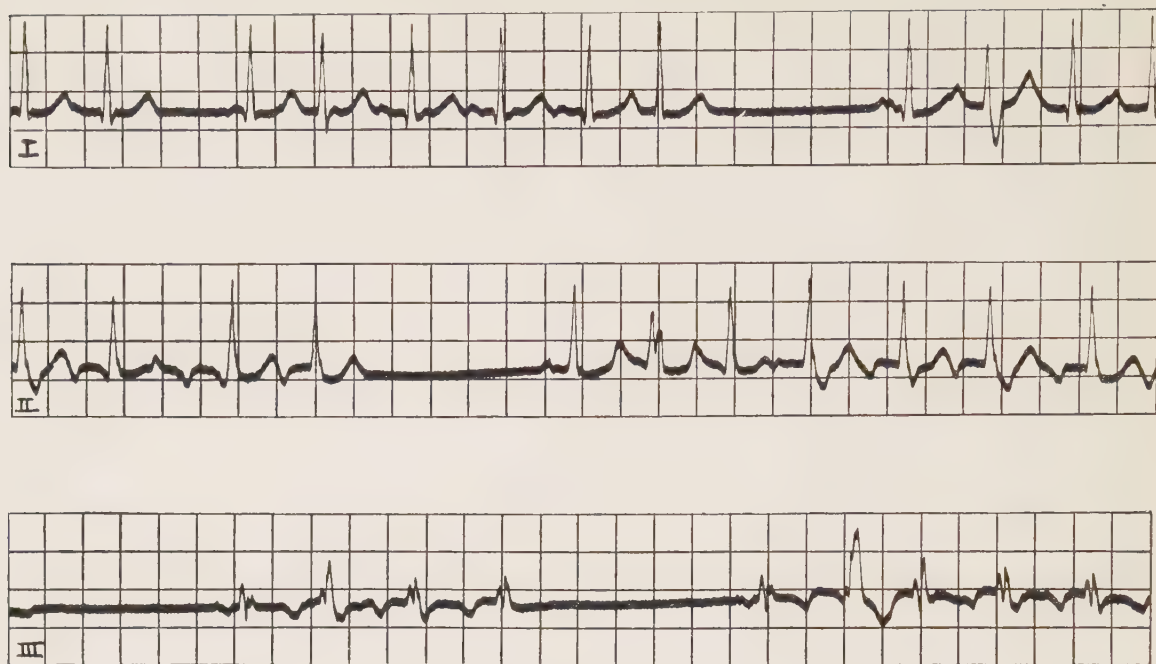


FIG. 74.—Record showing auricular flutter with 2 : 1 block interrupted by sino-auricular block. The auricular rate is 220, and the ventricular rate 107 contractions each minute during the periods of acceleration. Record obtained from a woman, aged sixty-six years, with hyperfunctioning adenomatous goiter.

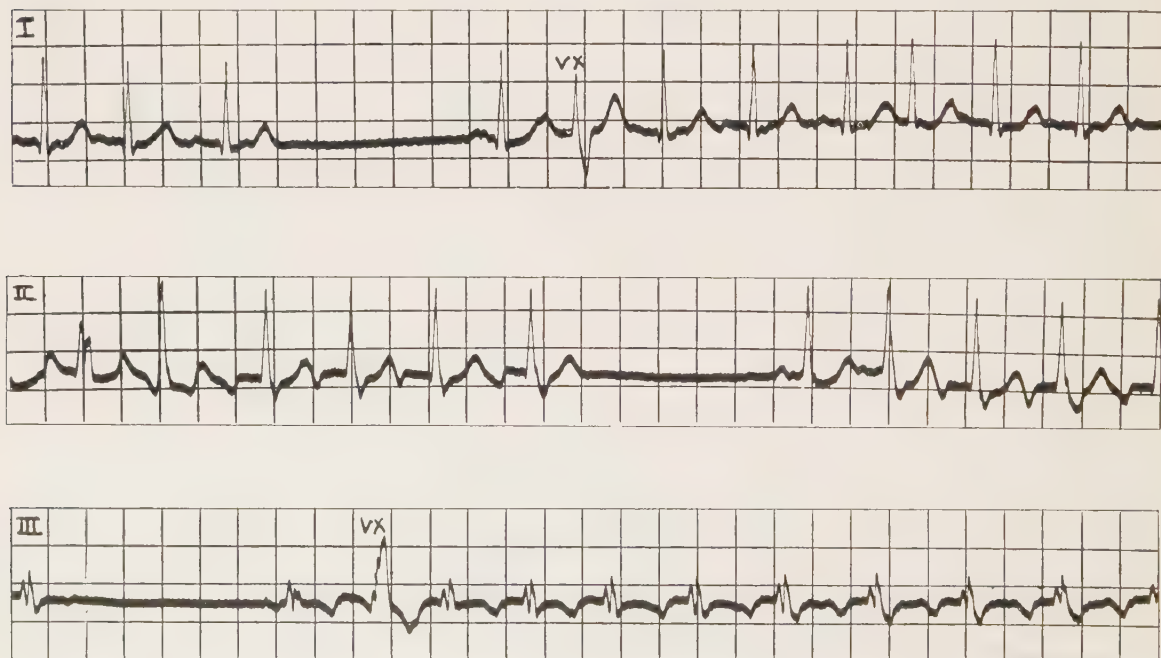


FIG. 75.—Record of the same patient who is represented in Figure 74, but taken on another day.

The electrocardiograms of auricular flutter are fairly characteristic, although at times variations in the records occur which may render their identification somewhat difficult. The most characteristic feature is the rapidity of the auricular deflections, P, displaying a

constant movement which results in a serrated graph. The individual P waves usually have a very angular appearance but the general contour often is somewhat rounded. The waves at times appear to be upright (positive) and again, in other records, downward (negative).

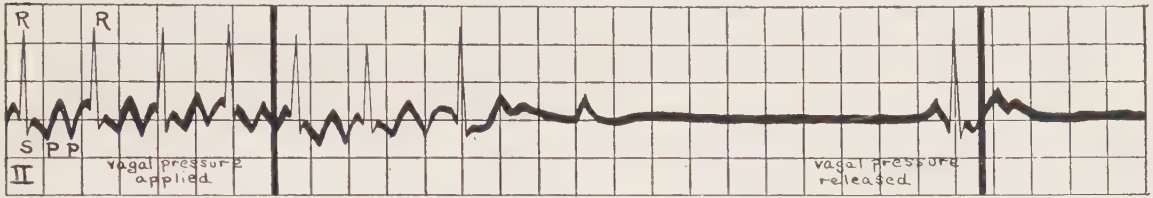


FIG. 76.—Record in lead II showing auricular flutter promptly arrested by right vagal pressure. Note the rather long period of cardiac asystole immediately following the termination of the flutter. Record obtained from a woman, aged fifty-five years, with hyperfunctioning adenomatous goiter.**

As has been stated, varying degrees of block usually occur in flutter, and the ventricle responds to every second, third, or fourth auricular impulse. In Figures 63, 64, 66, and 67 are shown typical examples of 2 : 1 flutter. The general similarity in the character and the contour of the P waves, especially in leads II and III, should be noted.

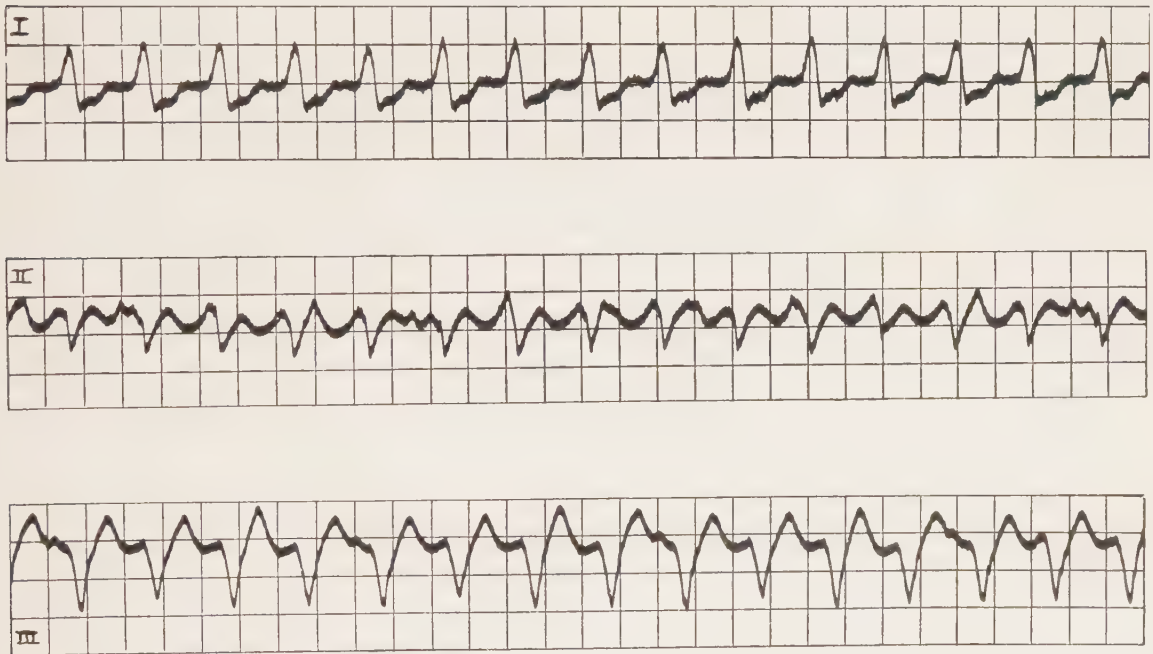


FIG. 77.—Record showing auricular flutter, with 2 : 1 and occasional 3 : 1 block, associated with disturbance in ventricular conduction as evidenced by the bizarre QRS complexes in all leads. Record obtained from a man, aged forty-four years, with hypertensive heart disease.

The true character of flutter is seldom clearly delineated in lead I; nevertheless, at times fairly definite changes are revealed, as seen in Figures 65 and 73. In Figures 65 and 68 are seen examples of 3 : 1 and 4 : 1 flutter. Although the general contour of the records is the same, slight individual variations in the degree of angulation of the P waves occur.

The T waves are usually not discernible, because they are usually submerged in the general alteration of the graph.

Variations in the degree of block from cycle to cycle are not uncommon, as shown in Figures 69 and 73, in which the association varies from 2 : 1 to 5 : 1. A rather unusual type of flutter is shown in Figures 70 to 72, in which occur variations in the contour and

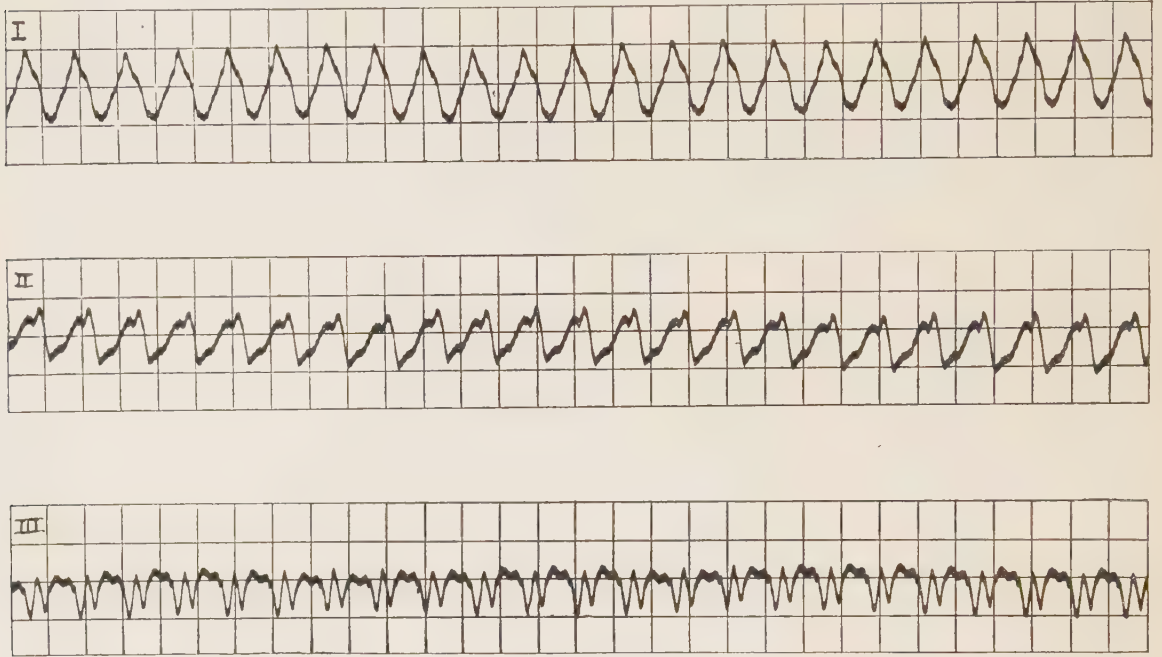


FIG. 78.—Record showing a rapid paroxysm of auricular flutter; the rate is 225 contractions each minute with a 1 : 1 association. Note the peculiar contour of the record which was obtained from a man, aged fifty-four years, with exophthalmic goiter.

in the amplitude of deflection of the P waves, associated with varying degrees of block. If only isolated complexes of this record were examined, considerable difficulty would be encountered in identifying it as flutter.

Flutter may be associated with other disturbances in cardiac mechanism. These are exemplified in Figures 74 and 75, in which irregular periods of sino-auricular block occur.

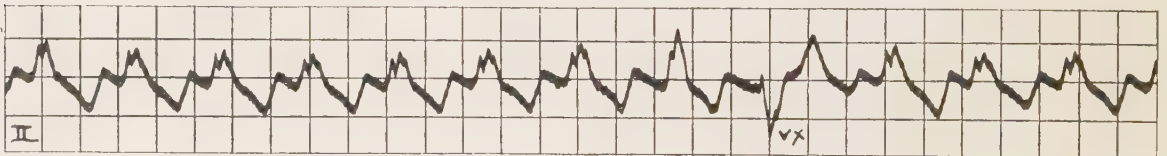


FIG. 79.—Record in lead II showing auricular flutter, with 2 : 1 block, associated with aberration of the QRS complexes. One premature ventricular contraction occurs in the record. The auricular rate is 254, and the ventricular rate 127 contractions each minute. Record of the same patient who is represented in Figure 78, but taken on another day.

In these records, also, the flutter mechanism is resumed at times by aberrant beats which possess the characteristics of premature ventricular contractions. The flutter complexes here are far from being typical and could readily lead to considerable difficulty in interpretation.

Paroxysmal flutter often behaves like other paroxysms of rapid heart action, and in Figure 76 is found an example of sudden termination of the paroxysm following the

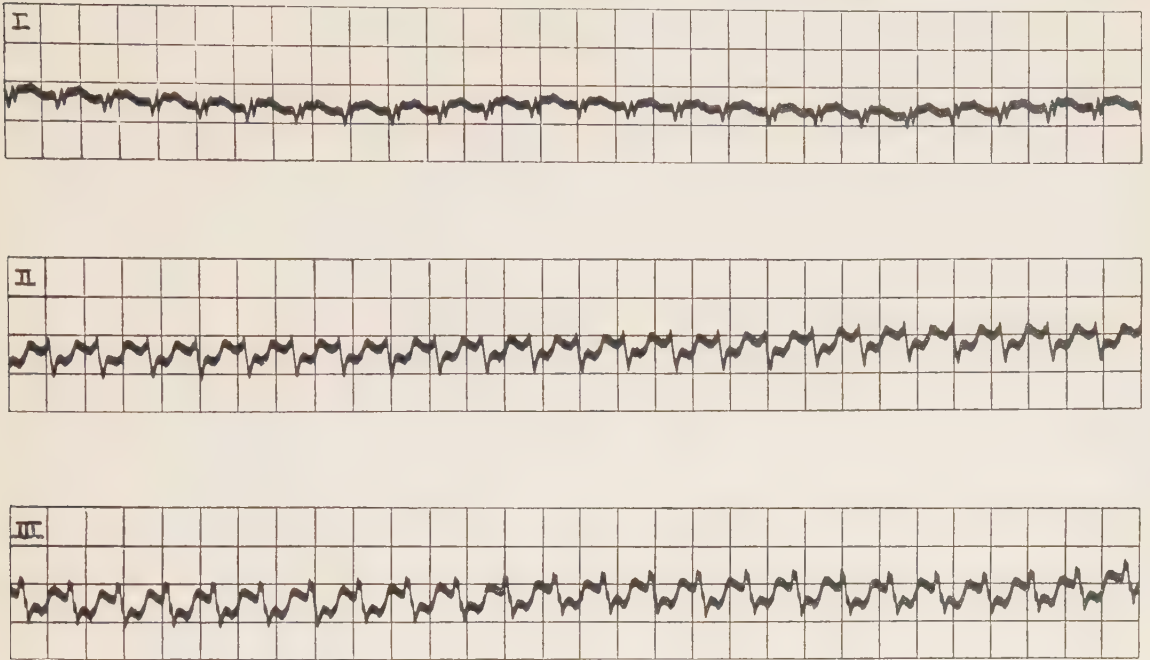


FIG. 80.—Record showing paroxysmal auricular flutter with 1 : 1 response. The rate is 240 contractions each minute. Record obtained from a man, aged forty-eight years, with recent myocardial infarction.

application of right vagal pressure. Note the relatively long period of cardiac asystole immediately following the arrest of the flutter mechanism.

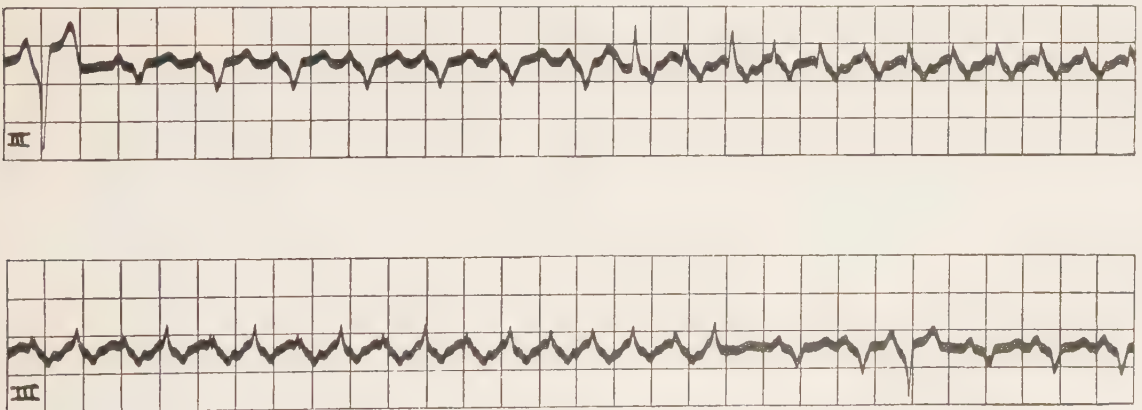


FIG. 81.—Two continuous records in lead III showing the onset of a paroxysm of tachycardia which apparently is auricular flutter. The rate during the paroxysm is 265 contractions each minute. Note the reversal in the direction of the waves. Occasional premature contractions occur. Record from a woman, aged thirty-four years, with cardiac hypertrophy of indeterminate origin.

Occasionally records of flutter are obtained which display aberrant ventricular contractions that are similar in form and duration to those associated with disturbed conduction in the ventricles (Figs. 77 and 79).

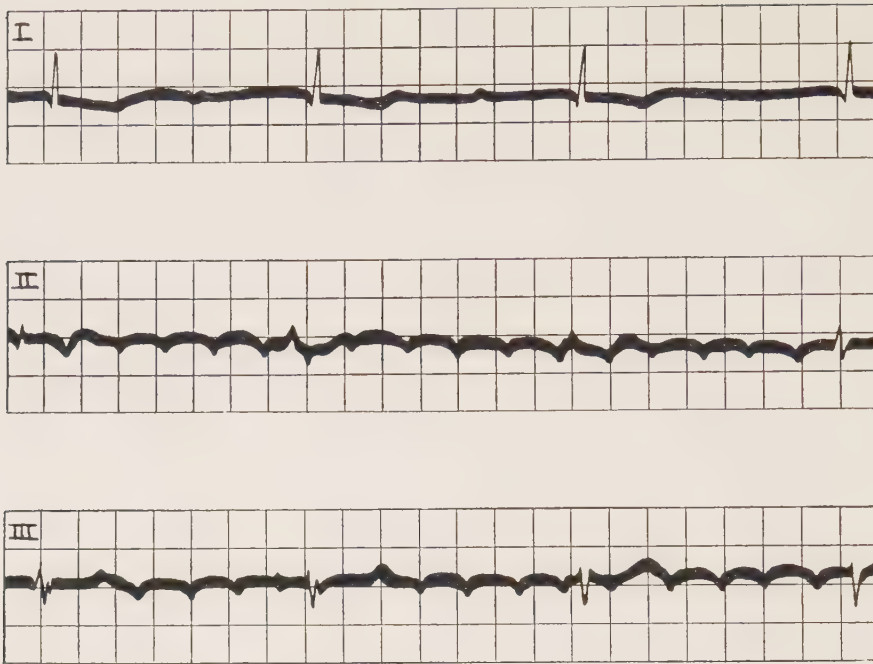


FIG. 82.—Record showing auricular flutter with established complete heart-block. Record obtained from a man, aged fifty years, with coronary and hypertensive heart disease.**

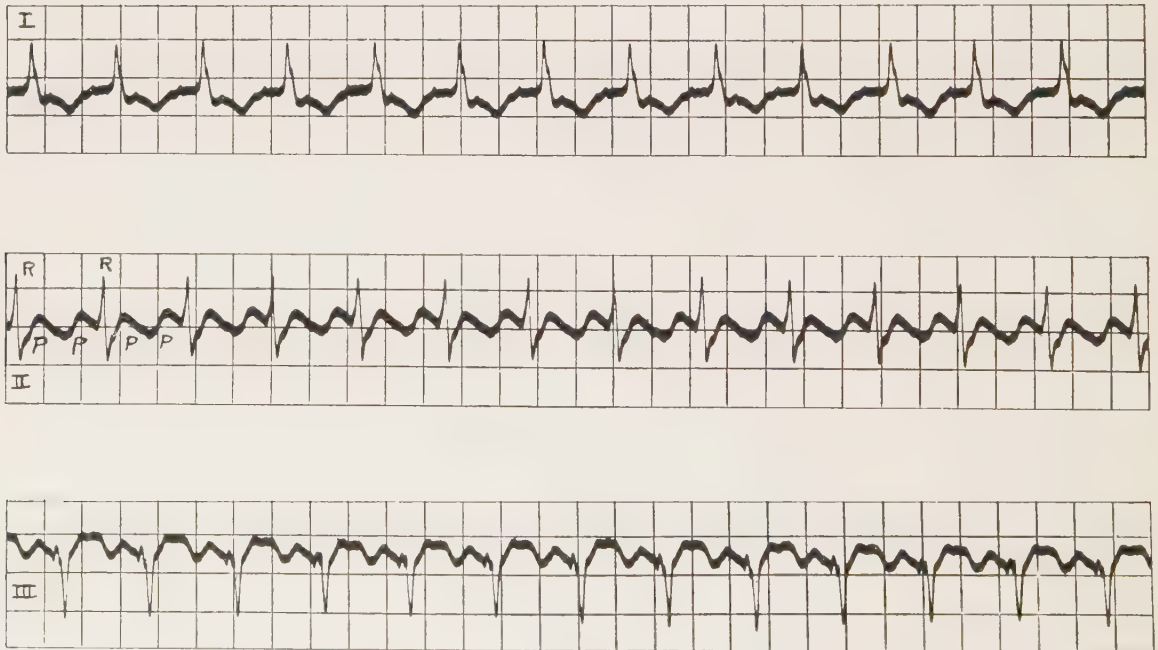


FIG. 83.—The three records of auricular flutter presented in Figures 83, 84, and 85 are from a man, aged fifty-eight years, with hypertensive heart disease. The records illustrate the results obtained from the employment of digitalis in treatment. The record, obtained on the day that treatment was instituted, shows auricular flutter with 2 : 1 block. The ventricular rate is 132, and the auricular rate 264 contractions each minute. Note the prominent, regularly spaced P waves especially noticeable in lead II. The T wave in lead I is inverted.

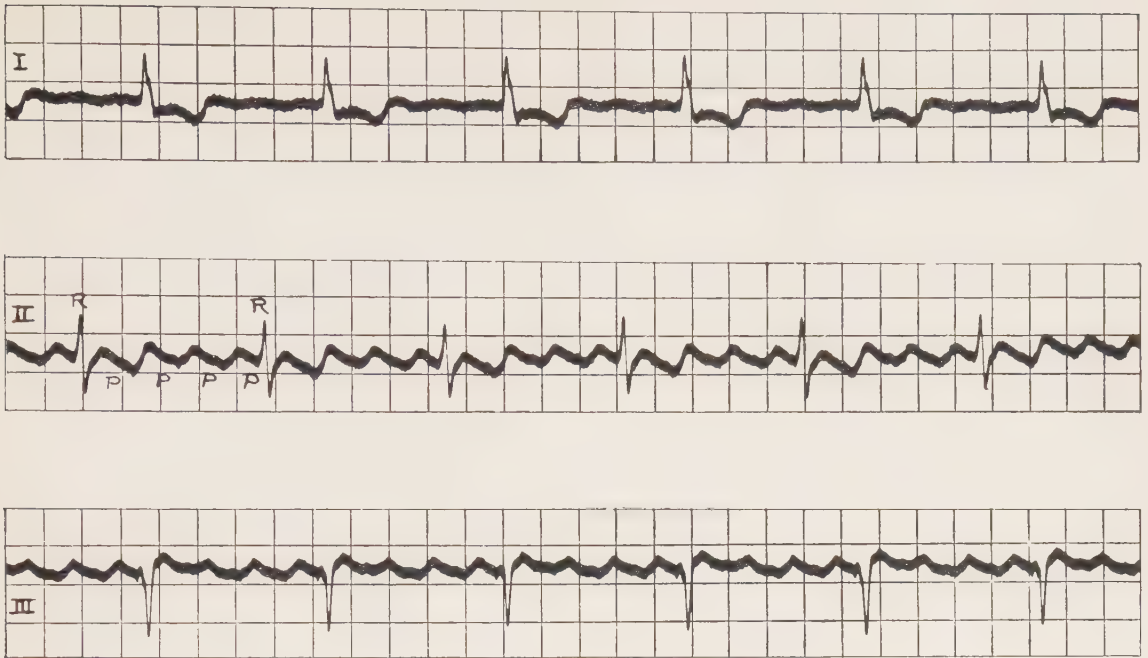


FIG. 84.—Record obtained thirteen days later (Fig. 83) showing auricular flutter with 4 : 1 block. The ventricular rate is 62, and the auricular rate 248 contractions each minute. The evidence of flutter is obtained entirely from leads II and III of this record. The T wave in lead I is inverted.

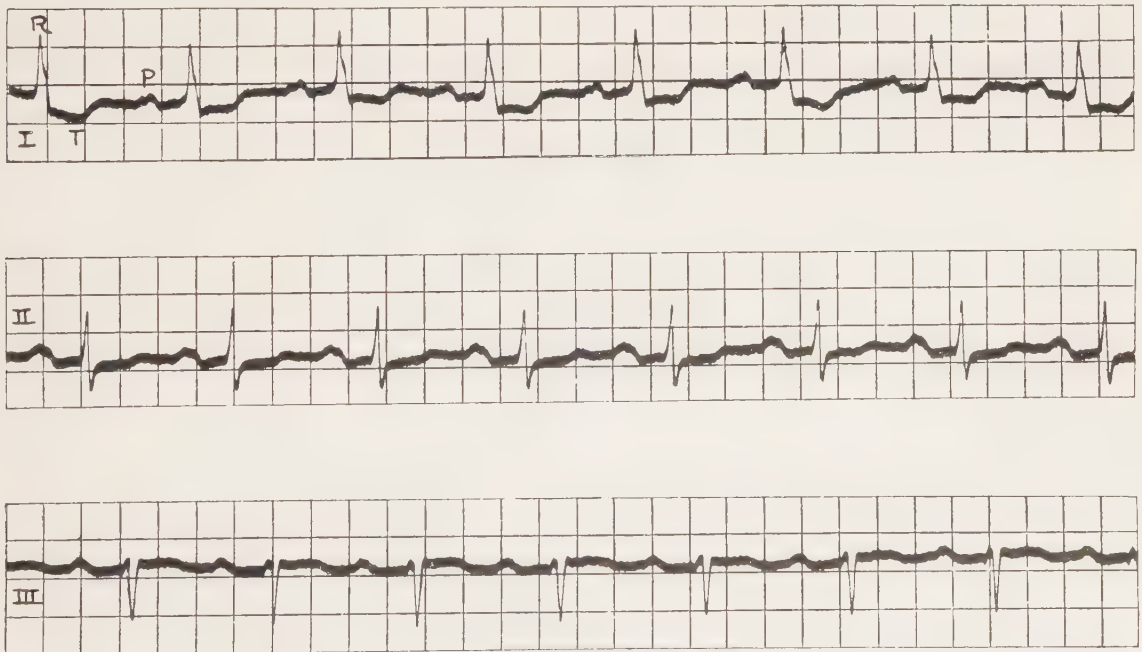


FIG. 85.—Record obtained three days later (Fig. 84) showing the restoration of normal rhythm. The T waves in leads I and II are diphasic and there is delay in auriculoventricular conduction; the P-R interval is 0.26 second. All records show preponderance of the left ventricle.

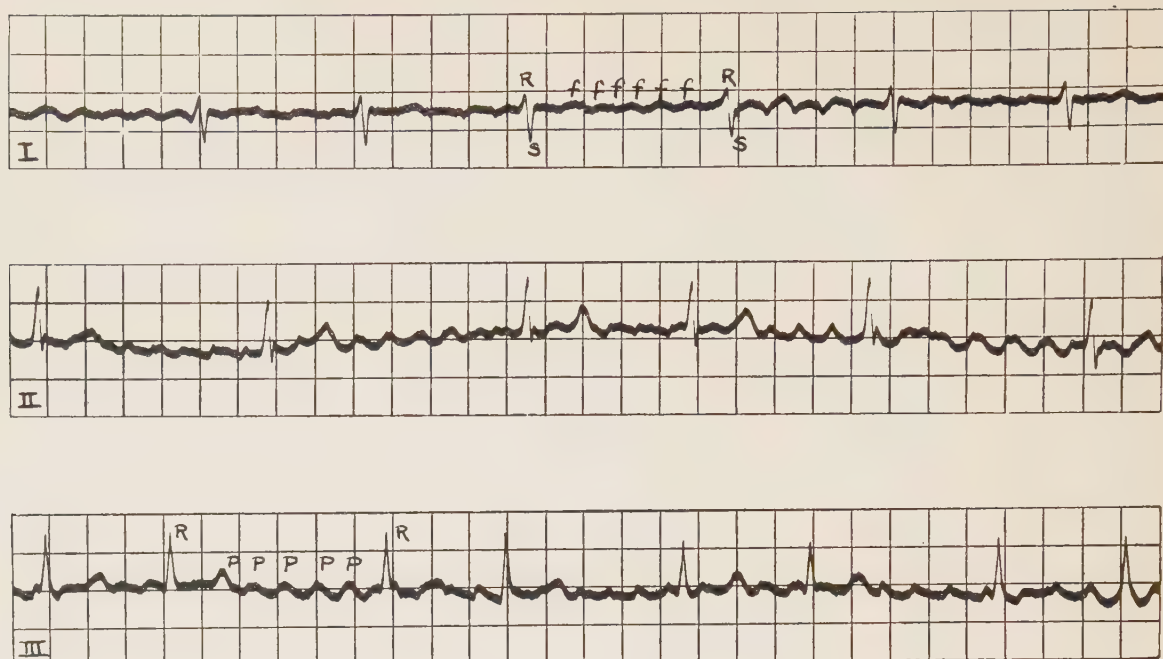


FIG. 86.—Record showing intermittent, coarse auricular fibrillation and impure auricular flutter. The differences between the fibrillary undulations and the irregular P waves are evident. Record obtained from a man, aged twenty-nine years, who had chronic rheumatic endocarditis with mitral stenosis.

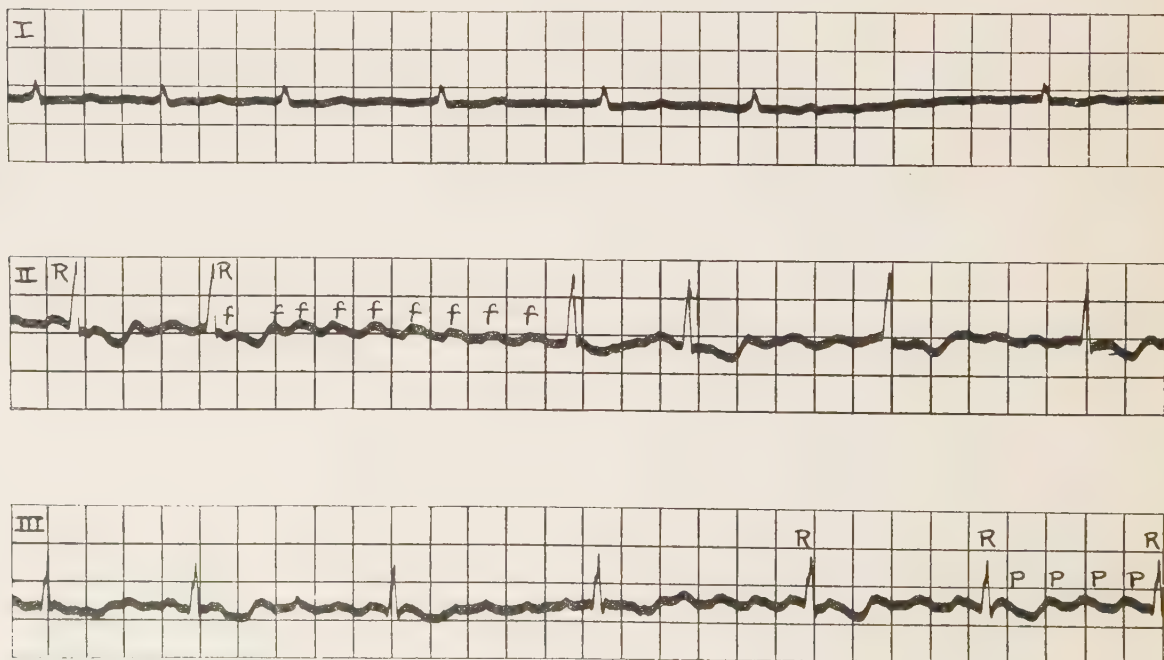


FIG. 87.—Record showing intermittent auricular fibrillation and impure auricular flutter. The QRS complexes in lead I are unusually low in amplitude, whereas slight notching of the ascending limbs occurs in lead III. Record obtained from a woman, aged thirty-eight years, with chronic rheumatic endocarditis with mitral stenosis.

Ventricular response to every auricular impulse (1 : 1 flutter) occasionally occurs; examples are found in Figures 78 and 80. The general character of these records differs materially from that of records already presented.

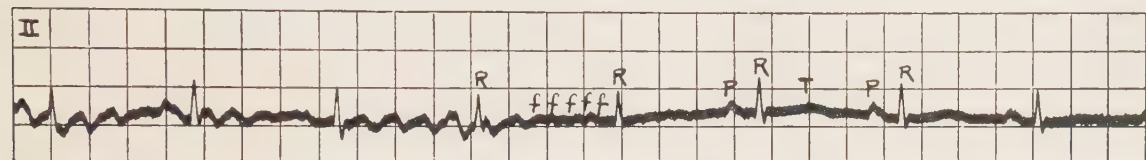
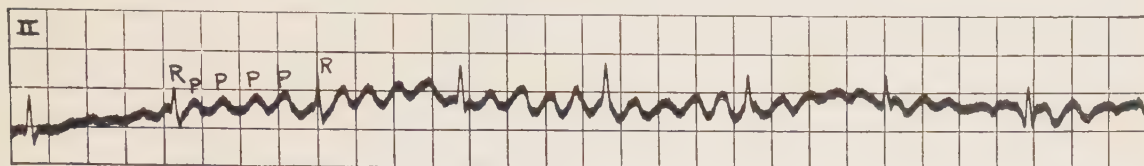


FIG. 88.—Continuous records in lead II showing intermittent auricular fibrillation and impure flutter and the restoration of normal rhythm. Record obtained from a man, aged forty-nine years, with syphilitic aortitis.

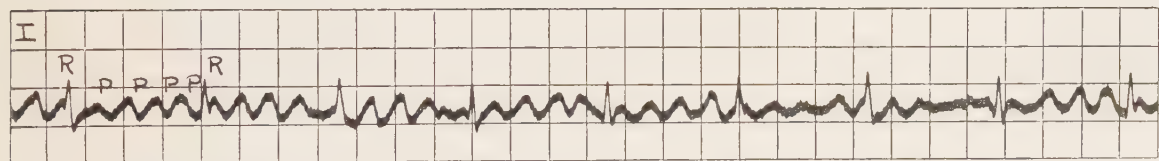
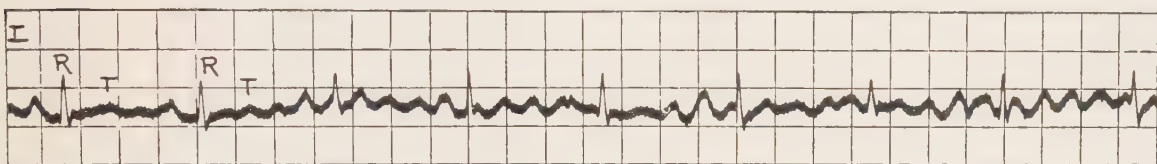


FIG. 89.—Record in lead I showing intermittent, coarse auricular fibrillation and impure flutter. The general tendency of the records is to exhibit waves resembling those of flutter, although less regularity in the undulations occurs. The two strips in this figure are continuous. Record obtained from a man, aged forty-nine years, with slight cardiac hypertrophy of indeterminate origin.

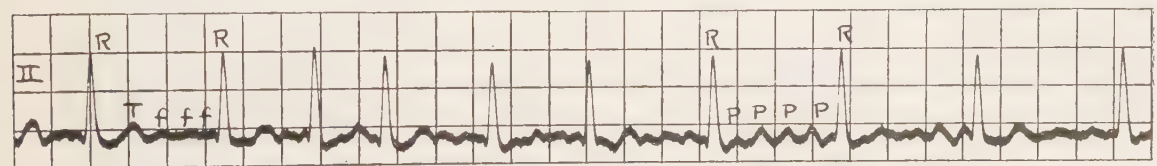


FIG. 90.—Record in lead II showing auricular fibrillation and intermittent, impure flutter. Flutter is well demonstrated in the complexes in the last half of the record. Record obtained from a man, aged forty-nine years, with cardiac hypertrophy associated with syphilitic aortitis.

Figure 81 is an interesting and unusual electrocardiogram which apparently is a record of flutter and displays an intermittent reversal in the direction of the waves. The exact interpretation of this record is somewhat uncertain.

Rarely, auricular flutter and established complete heart-block coexist. The record in Figure 82 is of the only case in my experience of this combination of abnormalities.

The arrest of auricular flutter by means of medication with digitalis is shown in Figures 83 to 85. The mechanism before treatment was 2 : 1 flutter. This soon was changed to 4 : 1 flutter and finally to normal sinus rhythm. Auricular fibrillation occurs before the return to normal rhythm. In the case presented this transitional mechanism was not detected, although unquestionably it was present.

Instances of impure flutter occurring with coarse auricular fibrillation are presented in Figures 86 to 90. Note the waves fairly characteristic of flutter in some cycles and the coarse, irregular fibrillary undulations in others. Records of this type clearly demonstrate the close relationship of auricular flutter and fibrillation.

Auricular flutter is not as uncommon as previously it was thought to be, and its more frequent recognition is attributable to the more general use of the electrocardiograph. Auricular flutter occurs in many different types of cardiac disease and is not characteristic of any particular one. It is probably most frequently observed in cases of mitral stenosis, especially when the lesion is well advanced, when considerable enlargement of the heart has occurred, and particularly when heart failure is present. In these cases it is prone to exist as a permanent mechanism until it is corrected by treatment. It is a rather common disturbance with exophthalmic goiter, in which it usually occurs as a paroxysmal phenomenon and very often terminates spontaneously. Flutter is not uncommon in hypertensive heart disease, during the state of failure, and is likely to alternate with auricular fibrillation. I have observed flutter in other cardiopathies; namely, adherent pericarditis, coronary disease, and occasionally in congenital disease of the heart.

I have never observed flutter if clear-cut evidence of organic heart disease was absent, and I doubt its occurrence as a purely neurogenic phenomenon. Recent statistical data regarding the prognosis in cases of flutter are not available to me at this time, but my impression is that the prognosis is dependent on the type, extent, and degree of the underlying cardiac disease, and on the thoroughness and effectiveness of treatment in abolishing the abnormal mechanism.

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Chapter VI

AURICULAR FIBRILLATION

The disordered action of the heart which is now known as auricular fibrillation was termed by early observers "pulsus irregularis perpetuus," "perpetual arrhythmia," and "mitral pulse." It constitutes about 50 per cent of the cases of persistent arrhythmia which are displayed by the human heart.

Lewis and his coworkers have demonstrated circus movement in clinical auricular fibrillation. A single circulating wave controls auricular activity. However, the wave

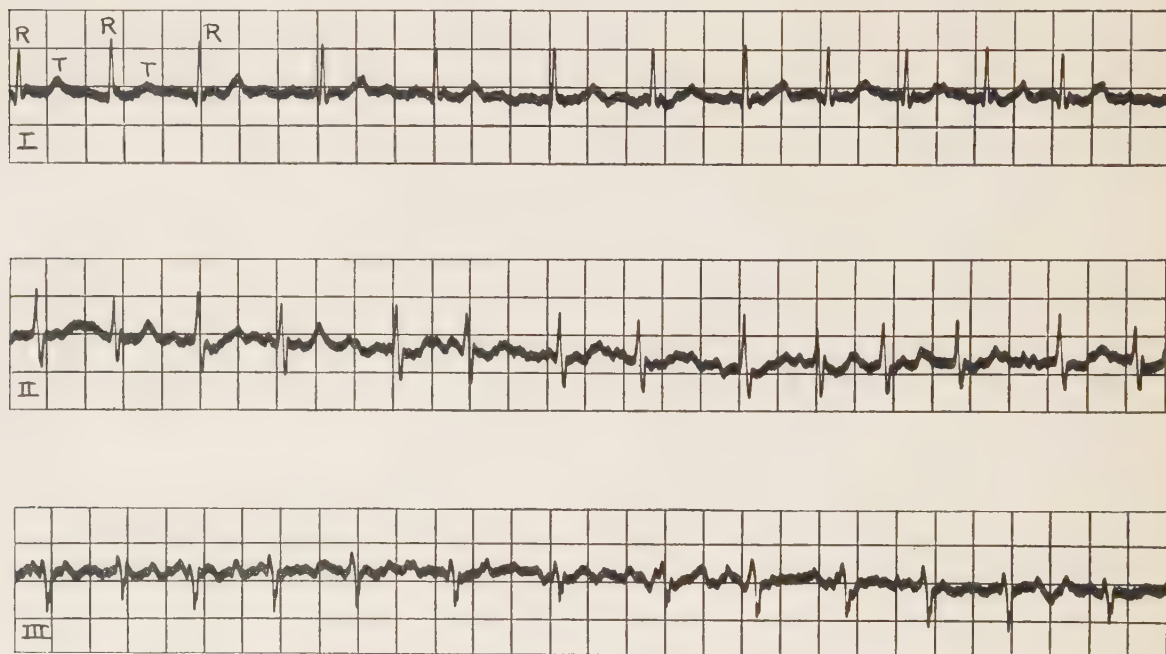


FIG. 91.—Record showing auricular fibrillation. The total irregularity, as well as the variation in amplitude of the R waves, is evident. Slight notching of the QRS complexes occurs in lead III and there is some preponderance of the left ventricle. Record obtained from a woman, aged sixty-four years, with hyperfunctioning adenomatous goiter.

does not pursue the fixed course that it does in flutter, as a change in the plane of movement occurs from time to time. The variations in this plane are attributed to the temporary establishment of new avenues of entry which are adopted by the wave of excitation and subsequent return of the wave to the original portal of entry. The mechanism of auricular fibrillation varies from cycle to cycle and from case to case, but it is fairly uniform in the same patient.

The electrocardiograms usually exhibit a total irregularity of ventricular complexes. The interval of occurrence of the ventricular complexes, which comprise the deflections QRS and T, continually varies. The ventricular complexes often do not differ in contour from those of normally acting hearts; yet they may present many individual variations.

Not infrequently the amplitude of the R wave varies from cycle to cycle, and this variation may be more pronounced in one lead than in the others. The P wave is absent, for dynamic contraction of the auricles has ceased. Small, rapid, irregular undulations, which must not be confused with P waves, frequently are present. They probably represent the fibrillary twitchings of the auricular musculature and the rate of discharge of stimuli

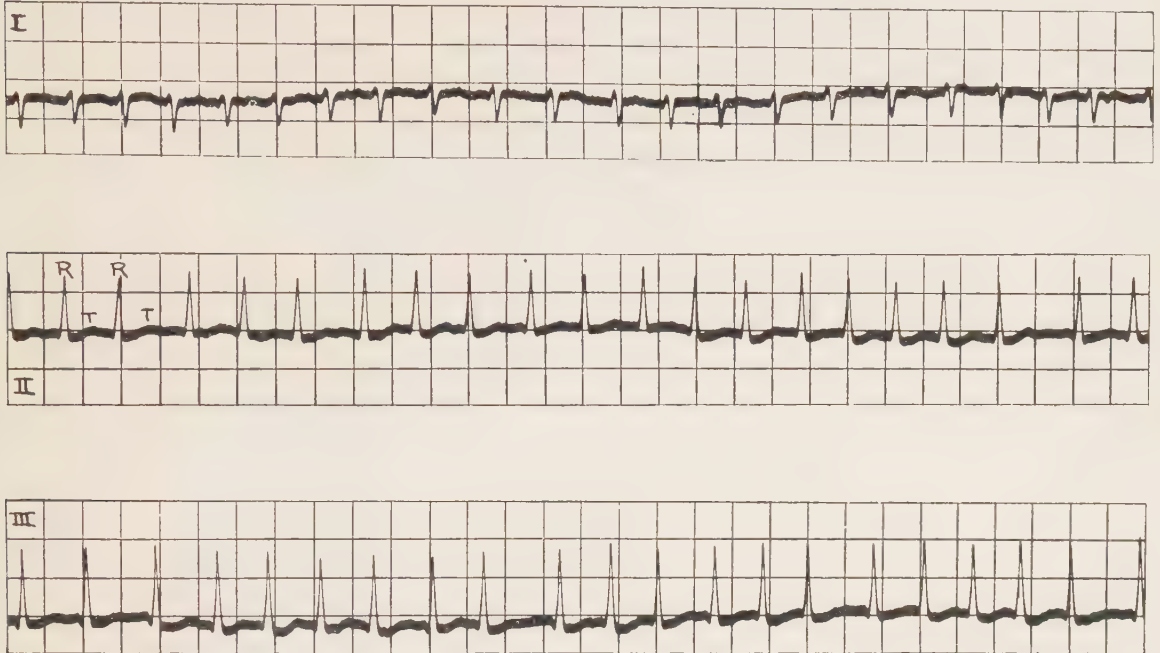


FIG. 92.—Record showing a paroxysm of auricular fibrillation; the rate is 200 contractions each minute. The total irregularity, as well as the variations of amplitude in the R waves, is readily seen. There is preponderance of the right ventricle. Record obtained from a man, aged fifty-three years, with exophthalmic goiter.

varies from 450 to 1000 a minute. These undulations, when clearly delineated, are designated by the symbol, *f*, and the electrocardiograms in which they appear have been described as representing fine or coarse auricular fibrillation, depending on the character of the fibrillary waves.

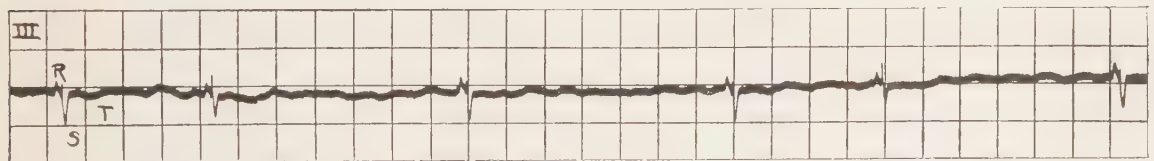


FIG. 93.—Record in lead III showing slow auricular fibrillation. The total irregularity, the variation in amplitude of the QRS complexes, and the fibrillary undulations are clearly shown. Record obtained from a woman, aged sixty-five years, with coronary sclerosis.

Auricular fibrillation may occur as a permanent, a transient, or a paroxysmal disorder of cardiac rhythm. The ventricular rates may be moderate, extremely rapid, or slow.

Examples of fine auricular fibrillation are shown in Figures 91, 92, 94, 95, 96, 98, 99, 102, 103, 105, 106, 108, 109, and 110; coarse auricular fibrillation is illustrated in Figures 93, 97, 100, 101, 104, 112, 113, 114, and 115.

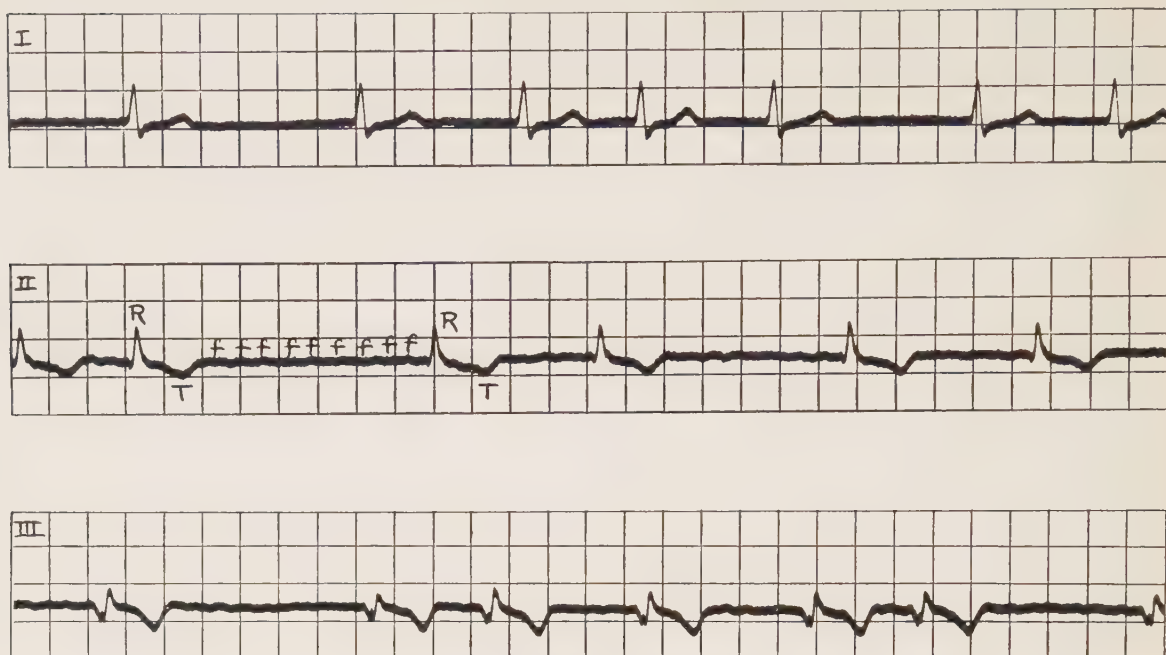


FIG. 94.—Record showing slow auricular fibrillation. The total irregularity is well marked and the fibrillary undulations are clearly shown in the long cycles. The T waves in leads II and III are inverted and are of increased amplitude. There is slight notching of the QRS complexes in lead III. Record obtained from a man, aged seventy years, with hypertensive heart disease and aortic sclerosis.

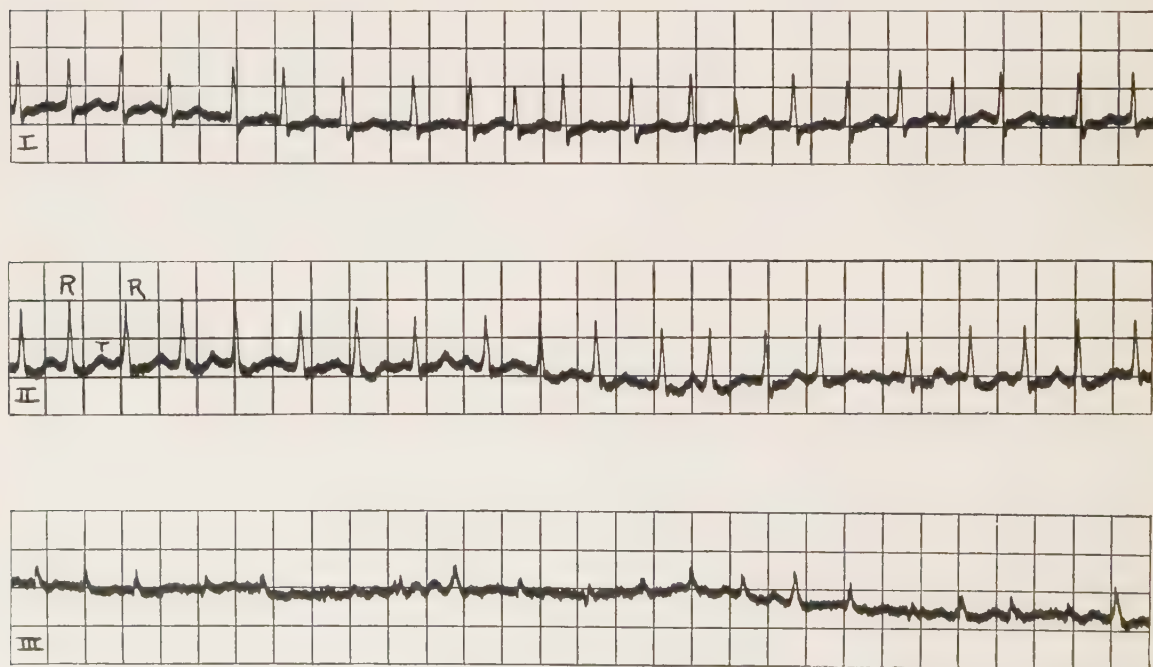


FIG. 95.—Record showing extremely rapid auricular fibrillation; the average rate is 192 contractions each minute. Even with this rapid ventricular action the total irregularity is evident and the variation in the amplitude of the R waves is definitely shown. Record obtained from a man, aged fifty-eight years, with hypertensive heart disease in the stage of congestive failure.

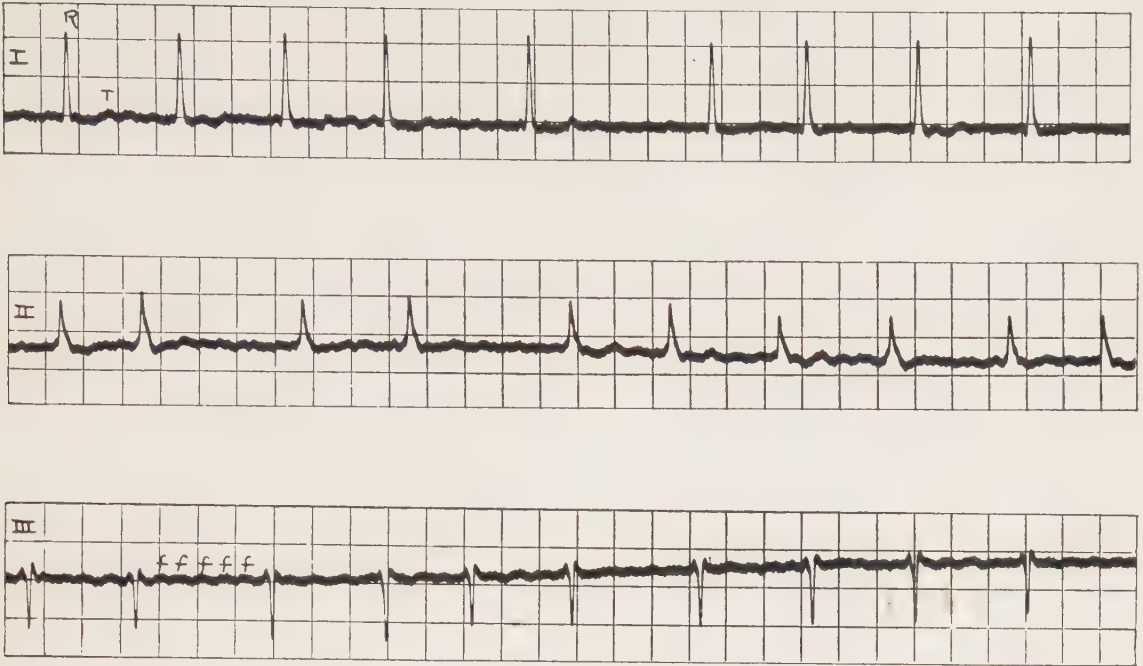


FIG. 96.—Record of auricular fibrillation clearly exhibiting the small, irregularly undulating fibrillary waves. There is variation in the amplitude of the R waves and total irregularity of rhythm. Left ventricular preponderance is present. Record from a man, aged sixty-three years, with hypertensive heart disease.

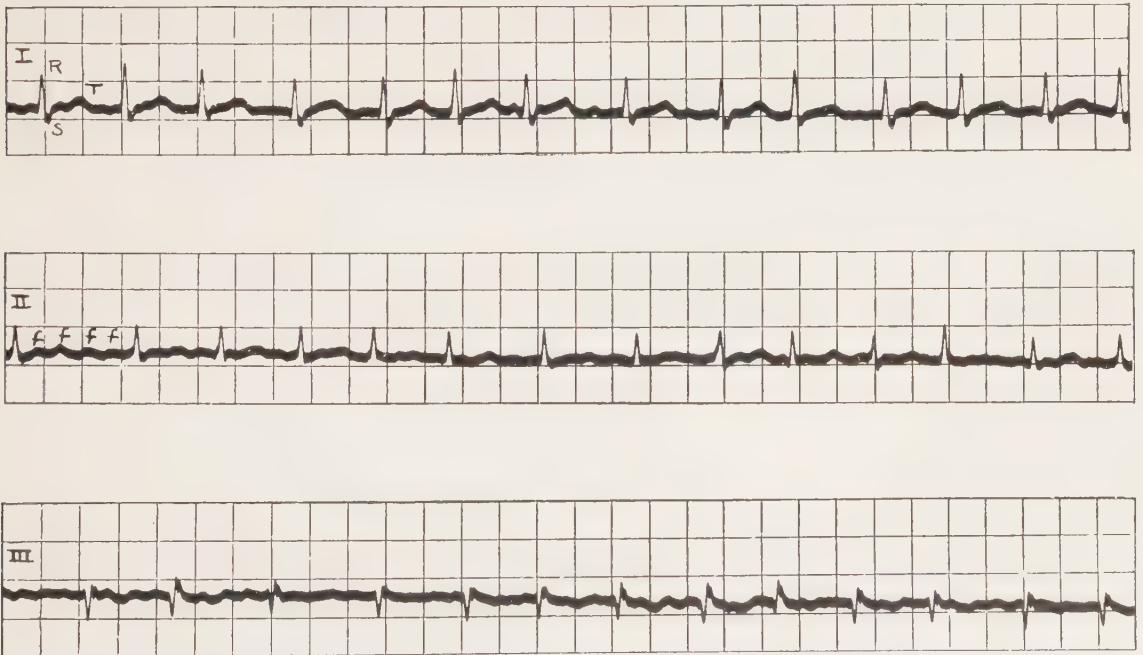


FIG. 97.—Record showing rapid auricular fibrillation; the rate is 132 contractions each minute. The total irregularity of beats is apparent and variation in the amplitude of the R waves occurs. Notching of the QRS complexes in lead III is present. Record obtained from a man, aged thirty-seven years, with hypertensive heart disease.

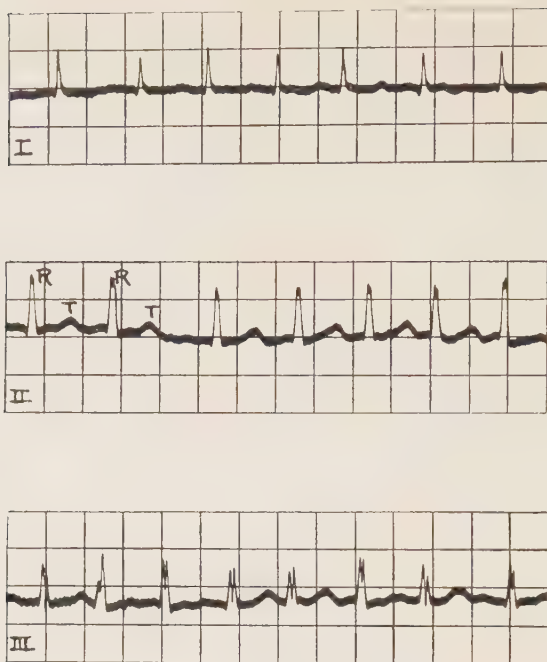


FIG. 98.—Record showing rapid auricular fibrillation. The total irregularity is apparent and considerable variations in the amplitude of the R waves occur. There is slight notching of the apex of the QRS complexes in lead II and marked notching, of varying form, in lead III. The latter complexes, to a large extent, resemble the so-called "M" complexes. Record obtained from a woman, aged sixty-three years, with hyperfunctioning adenomatous goiter and essential hypertension.

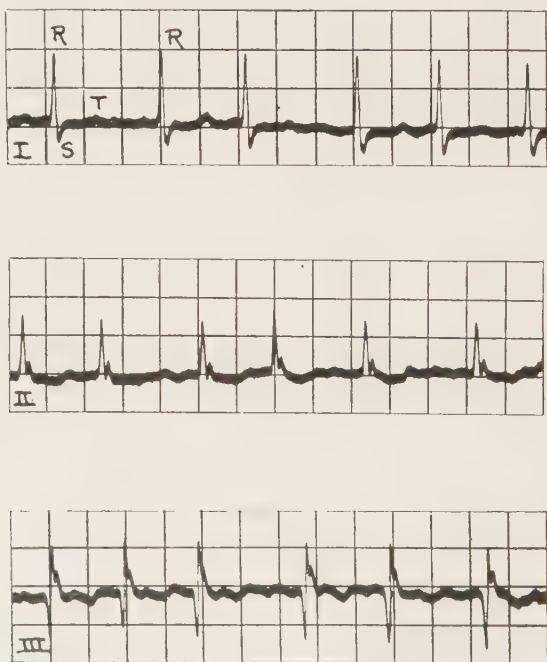


FIG. 99.—Record showing rapid auricular fibrillation and exhibiting notching of the descending limbs of the QRS complexes in leads II and III. Note the constancy of this notching. Record obtained from a woman, aged fifty-eight years, with hyperfunctioning adenomatous goiter.

Rapid paroxysmal auricular fibrillation is shown in Figures 92 and 95. The onset of a paroxysm is seen in Figure 108, and in Figure 109 is a record of the termination of a paroxysm of rapid auricular fibrillation.

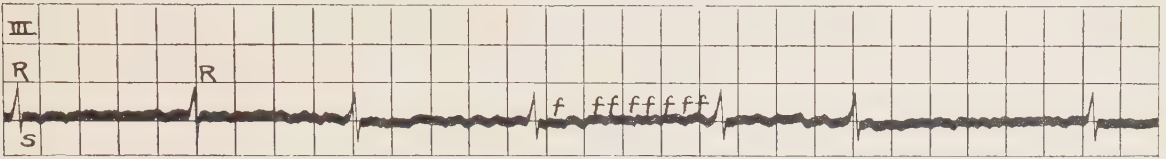


FIG. 100.—Record in lead III showing auricular fibrillation. The total irregularity is apparent and the fibrillary undulations are well marked in all cycles. Record obtained from a woman, aged sixty-four years, with exophthalmic goiter and congestive heart failure.

It is not unusual to see the arrhythmia irregularly interrupted by premature ventricular contractions which result from different foci of irritability (Figs. 101 to 107). In Figures 105 and 106 premature ventricular contractions occur alternately with the dominant ventricular rhythm of fibrillation.

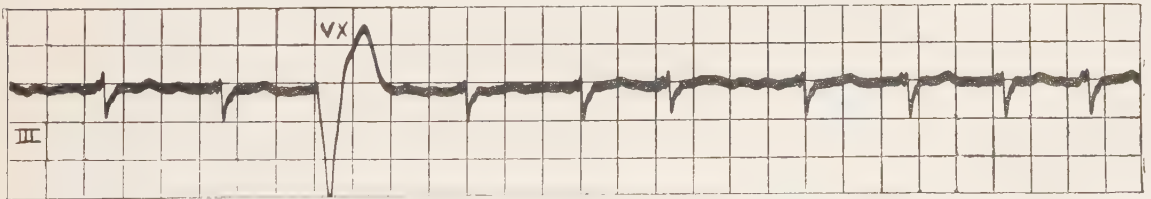
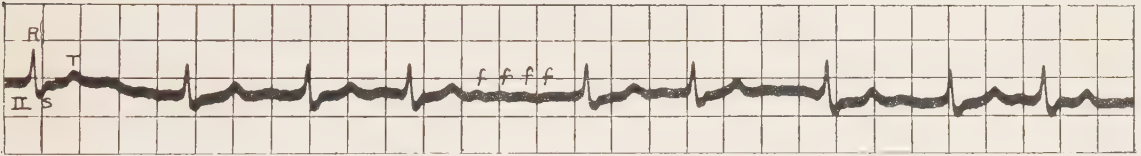
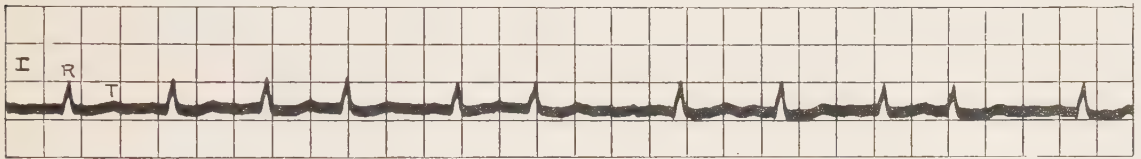


FIG. 101.—Record showing auricular fibrillation with premature ventricular contractions. The rhythm is totally irregular and some variation in the amplitude of the R waves is present. Fibrillary undulations are seen in the long cycles. Slurring of the QRS complexes occurs in leads I and III, with slight notching of the ascending limbs of the complexes in lead III. Note the extremely bizarre contour, with its high amplitude, of the premature contraction present in lead III. There is preponderance of the left ventricle. Record obtained from a man, aged sixty-seven years, with hypertensive and coronary heart disease.

Occasionally, the ventricular action in auricular fibrillation is rhythmic, especially in cases with slow rates (Fig. 110).

Other associated abnormalities occur, and occasionally auricular fibrillation is asso-

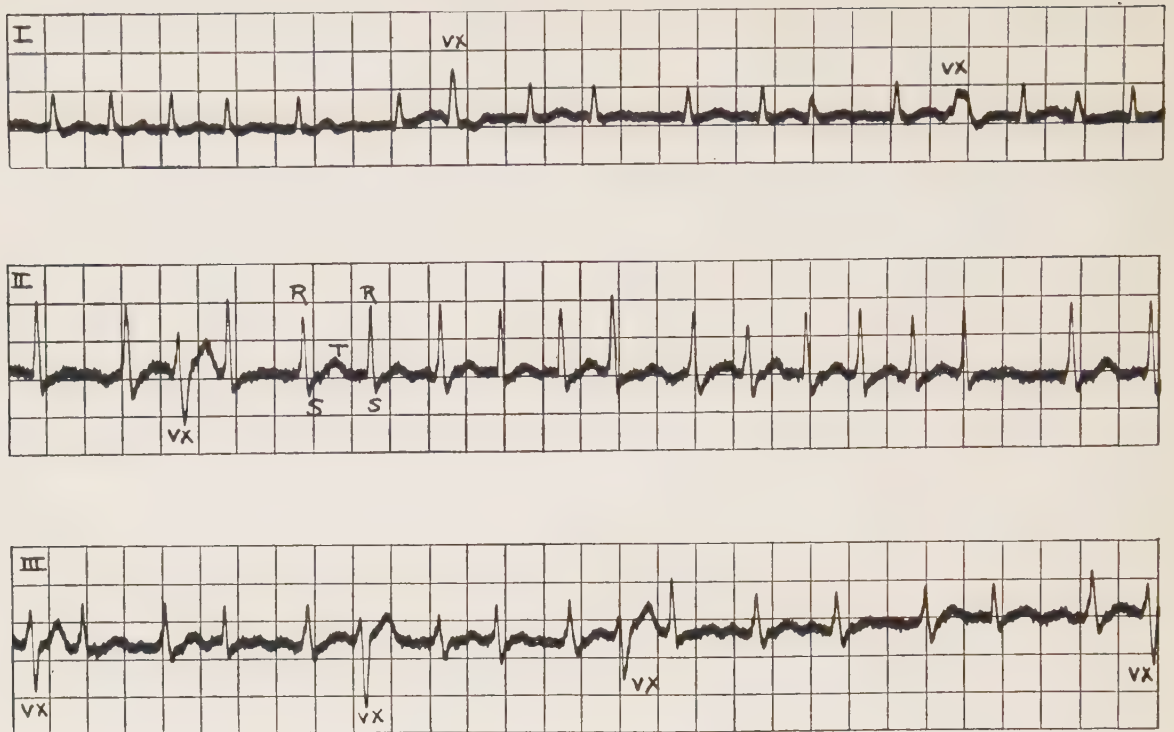


FIG. 102.—Record showing auricular fibrillation with premature ventricular contractions. A total irregularity occurs independent of the premature interruptions. Considerable variations in the amplitude of the R waves occur. Record obtained from a man, aged forty-four years, with hyperfunctioning adenomatous goiter.

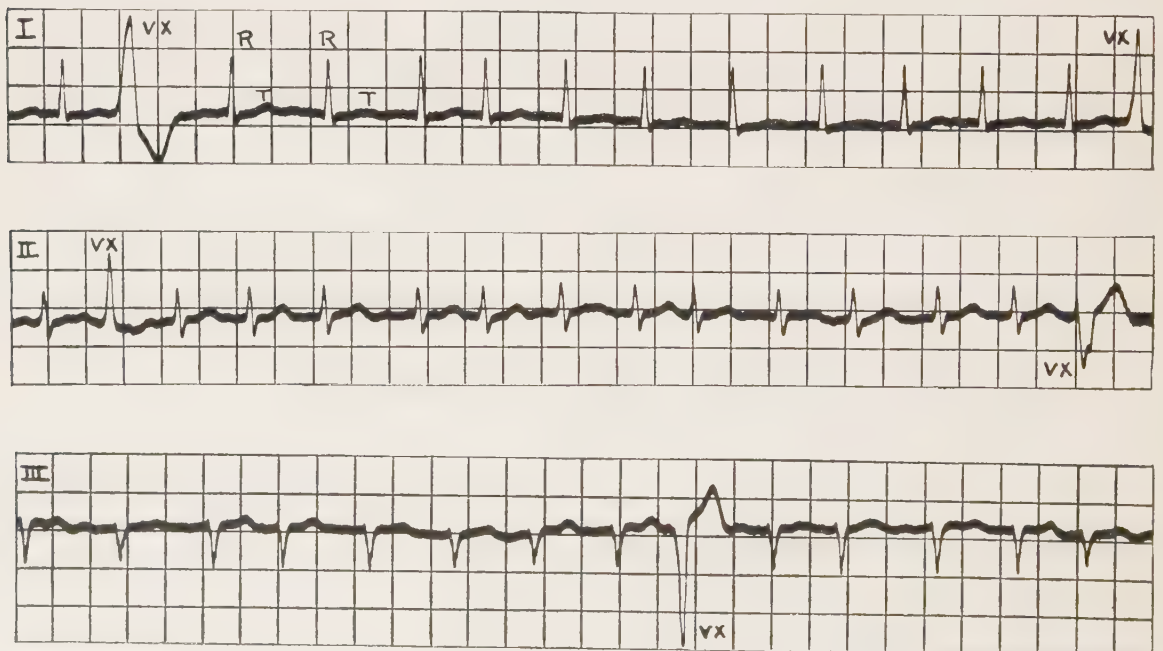


FIG. 103.—Record showing auricular fibrillation with premature ventricular contractions arising in different regions. The total irregularity of rhythm is apparent and considerable variation in the amplitude of the R waves occurs. Record obtained from a woman, aged sixty-eight years, with hyperfunctioning adenomatous goiter.

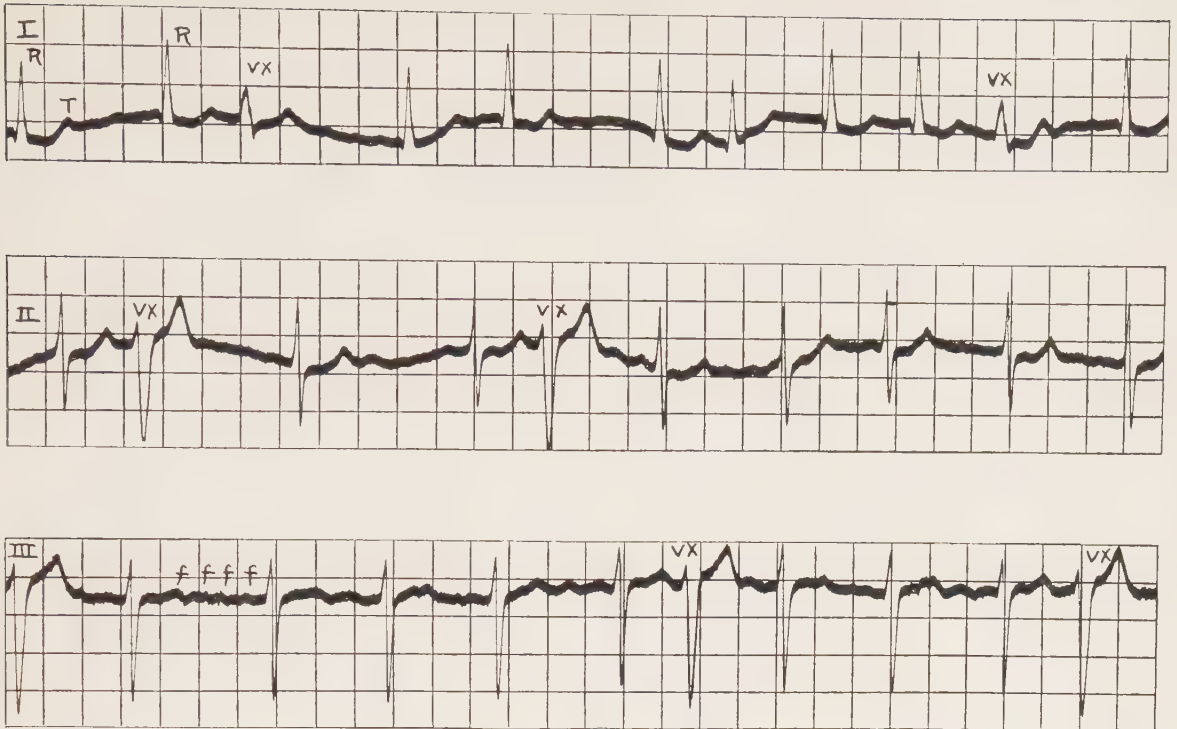


FIG. 104.—Record showing auricular fibrillation with premature contractions arising in the left ventricle. A total irregularity of ventricular action occurs independent of the interruptions. Variation in the amplitude of the R waves is apparent and fibrillary undulations are seen in some of the longer cycles. There is rather marked preponderance of the left ventricle. Record obtained from a woman, aged sixty-seven years, with hyperfunctioning adenomatous goiter.

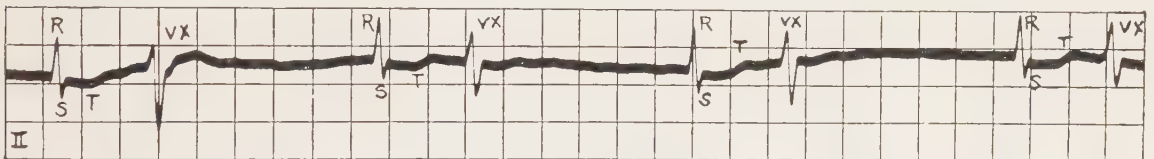


FIG. 105.—Record in lead II showing auricular fibrillation, with alternating premature ventricular contractions of slightly varying form. Record obtained from a woman, aged thirty-eight years, who had chronic rheumatic endocarditis with mitral stenosis.

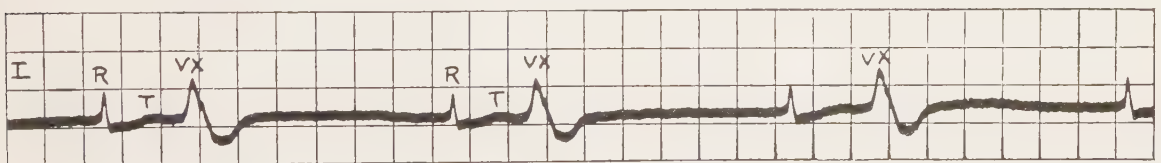


FIG. 106.—Record in lead I showing auricular fibrillation with alternating premature ventricular contractions. Note the unusual regularity of the premature contractions. Record obtained from a man, aged fifty years, with hypertensive heart disease.

ciated with established complete heart-block, as seen in Figures 111 and 112. In the case represented in Figure 113 transient, complete auriculoventricular dissociation occurred. Associated disturbance in ventricular conduction is not unusual in auricular fibrillation, as shown in Figures 110, 111, and 113.

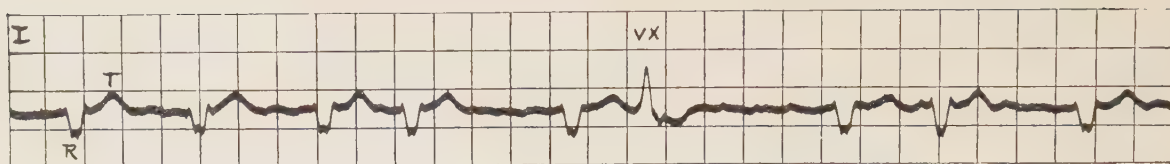


FIG. 107.—Record in lead I showing auricular fibrillation with a premature ventricular contraction. Note the abnormal character of the QRS complexes of the dominant arrhythmia; the base width of the complexes represents 0.12 second and definite notching occurs. Record obtained from a man, aged forty-eight years, who had hypertensive heart disease with heart failure.

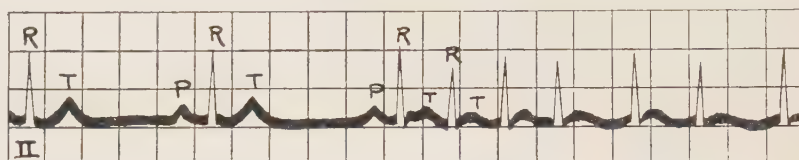


FIG. 108.—Record in lead II showing the onset of a paroxysm of auricular fibrillation. The total arrhythmia and the variation in amplitude of the R waves are apparent. The P waves are absent. Record obtained from a woman, aged twenty-eight years, with chronic rheumatic endocarditis with mitral stenosis.**

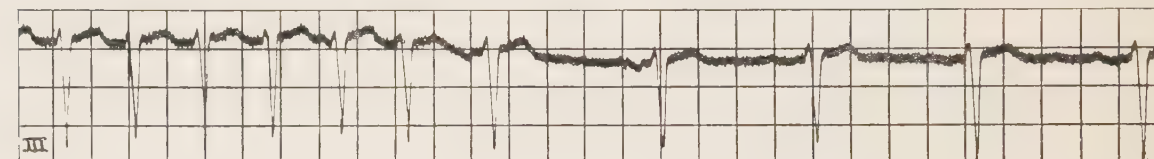
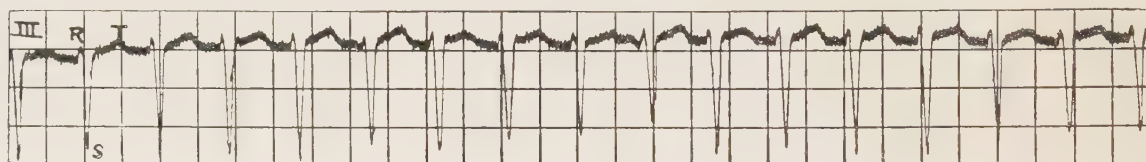


FIG. 109.—Record in lead III showing the termination of a paroxysm of auricular fibrillation; the rate varies from 167 to 63 contractions each minute. The total irregularity and the variation in the amplitude of the S waves are apparent. Record obtained from a man, aged fifty-eight years, with hypertensive heart disease.

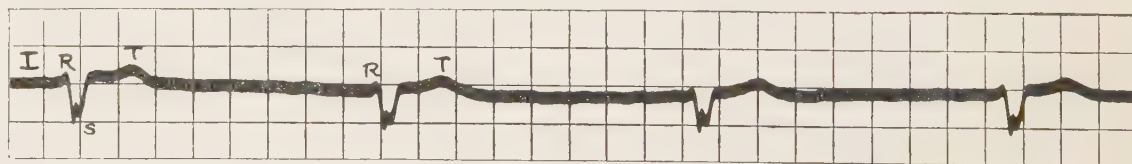


FIG. 110.—Record in lead I showing auricular fibrillation with regular ventricular action. The QRS complexes are notched and directed downward. Record from a man, aged sixty-six years, with hypertensive and coronary heart disease.

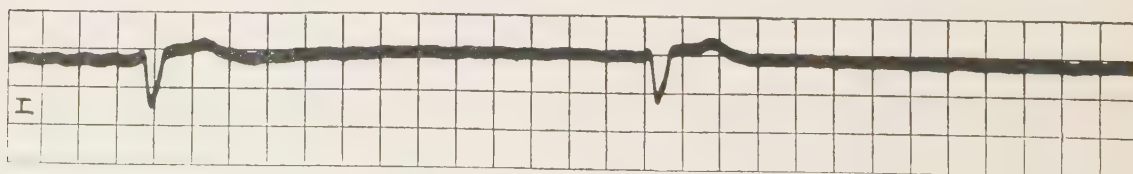


FIG. 111.—Record in lead I showing auricular fibrillation with complete heart-block. The QRS complexes are bizarre. The rate is 39 contractions each minute. Record from a patient, aged sixty-seven years, with coronary sclerosis.

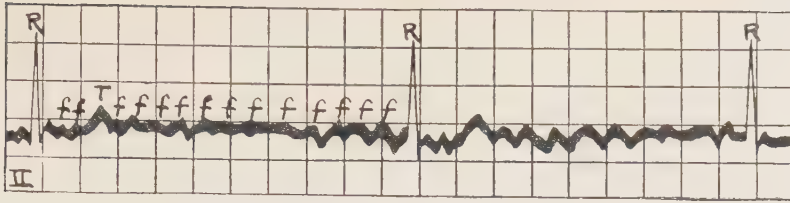


FIG. 112.—Record in lead II showing auricular fibrillation with complete heart-block. Note the coarse, fibrillary undulations. Record obtained from a man, aged fifty-four years, with coronary sclerosis.**

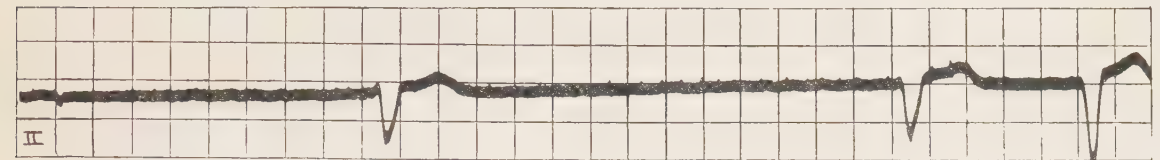
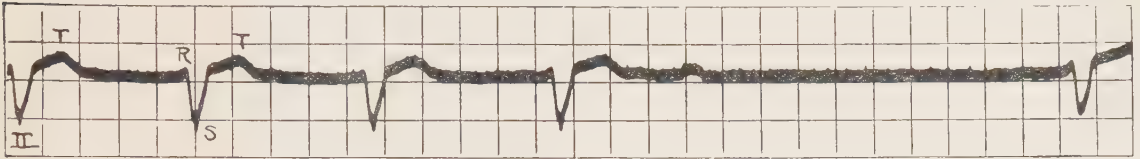


FIG. 113.—Record in lead II showing auricular fibrillation and transient complete auriculoventricular dissociation. The QRS complexes are bizarre, the intervals averaging 0.12 second, and some changes in contour of the individual complexes are apparent. Records are continuous, and are from a man, aged sixty-two years, with coronary sclerosis.

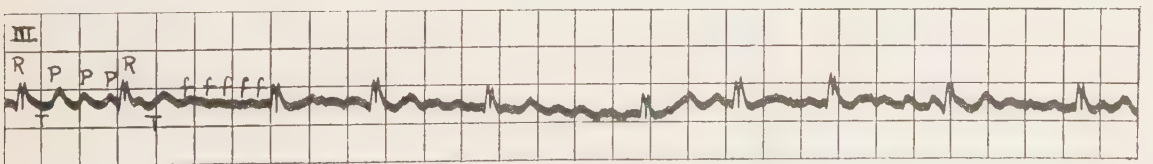
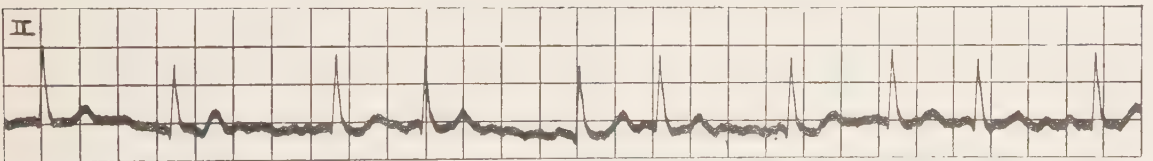
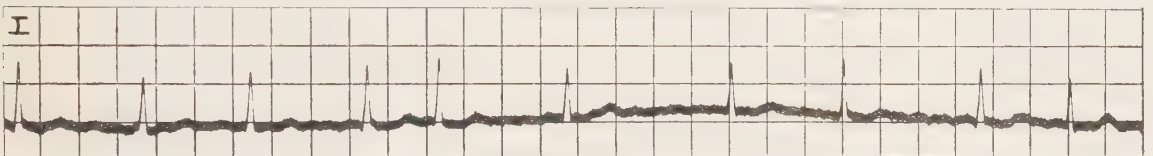


FIG. 114.—Record showing intermittent, coarse auricular fibrillation and impure auricular flutter. The differences between the coarse, fibrillary undulations and the irregular P waves are clearly seen. Record obtained from a man, aged fifty-nine years, with hyperfunctioning adenomatous goiter.

As emphasized in the foregoing chapter, the coexistence of auricular fibrillation and impure flutter is not unusual; examples are found in Figures 114 and 115.

Auricular fibrillation may occur in any type of heart disease, although it occurs more frequently in certain forms. Like auricular flutter, it probably is observed most frequently in cases of mitral stenosis. It is often associated with heart failure, although it does occur when the heart apparently is relatively efficient. Not uncommonly, it first appears coincidentally with the onset of heart failure. Hypertensive heart disease, exophthalmic goiter, and hyperfunctioning adenomatous goiter frequently are accompanied by auricular fibrillation. In the group associated with goiter it may occur in an intermittent or paroxysmal manner and may disappear spontaneously after the cessation of hyperthyroidism. Its occurrence in coronary disease is more common than formerly it was believed to be. Auricular fibrillation rarely is observed in aortic disease and, when present, it is apparently of considerable prognostic significance. It has been observed in adherent pericarditis, in congenital heart disease, and in mechanical interference with cardiac activity such as that which is observed in pronounced instances of kyphoscoliosis. That the association of subacute bacterial endocarditis and auricular fibrillation is extremely unusual is of interest.

Some patients may have auricular fibrillation for many years, apparently without material interference with their efficiency or happiness; yet, in many instances, death

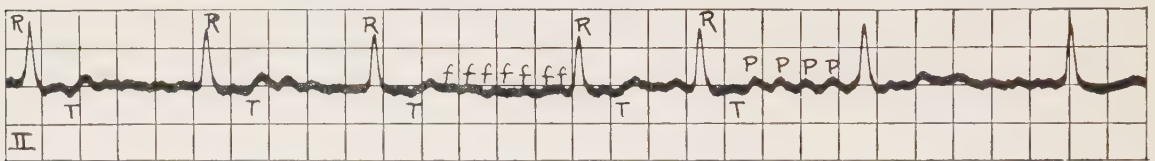


FIG. 115.—Record in lead II showing intermittent, coarse auricular fibrillation and impure flutter. The total irregularity is apparent, and the differences between the fibrillary undulations and the irregular P waves are clearly shown. Record obtained from a man, aged thirty years, who had chronic rheumatic endocarditis with mitral stenosis and insufficiency.

from heart failure occurs within a relatively short time after the inception of the arrhythmia. I have never observed a case in which purely neurogenic influences have caused auricular fibrillation. As yet, constant histopathologic changes have not been found in hearts which in life exhibited auricular fibrillation. Prognosis in heart disease embodies many factors, the most important of which are the type of a cardiopathy, the degree and extent of involvement, the inherent tendency of the lesion to progress, and the adequacy of the instituted regimen. Whether auricular fibrillation itself is an important factor in prognosis is doubtful; yet it is interesting to study mortality statistics of persons who had this disturbed cardiac mechanism. Statistical studies of this condition are reliable only within certain limits because of the great differences in the degree of cardiac involvement represented in the individual patients of a series and because of the obvious fact that the character of the series as studied in various localities will be variable.

In a group of 392 patients with auricular fibrillation which occurred in different forms of heart disease, it was found that 152 patients (39 per cent) had died as the result of cardiac disease within an average of eleven months following examination (Table 3). In analyzing this group of cases further, with reference to the type of lesion, some rather interesting data were elicited.

TABLE 3
MORTALITY IN CASES OF AURICULAR FIBRILLATION WITHOUT REGARD TO TYPE OF LESION

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
10-19	3	1	2	1	33	38		2	67
20-29	29	19	10	13	45	14	3	13	45
30-39	58	28	30	27	47	11	2	29	50
40-49	104	46	58	34	33	11	3	67	64
50-59	126	63	63	42	33	10	3	81	64
60-69	58	33	25	29	50	11	5	24	41
70-79	14	12	2	6	43	11	4	4	21
Total and average.	392	202	190	152	39	11	20	220	56

Of ninety-eight patients with mitral stenosis and auricular fibrillation, fifty (51 per cent) died of heart disease within an average of eleven months following examination (Table 4). The rather unusual association of aortic disease and auricular fibrillation has

TABLE 4
MORTALITY IN CASES OF AURICULAR FIBRILLATION AND MITRAL STENOSIS

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
10-19	3	1	2	1	33	38		2	67
20-29	15	11	4	8	53	12		7	47
30-39	28	17	11	15	54	7		13	46
40-49	29	15	14	14	48	15		15	52
50-59	14	7	7	5	36	15	1	8	64
60-69	8	5	3	6	75	6		2	25
70-79	1	1		1	100	14			
Total and average.	98	57	41	50	51	111	1	47	48

already been mentioned, and in Table 5 are the data based on only ten cases. Four patients (40 per cent) died of heart disease within an average of seven months following examination.

Sixty-nine patients with hypertensive heart disease associated with auricular fibrillation were traced; thirty-five (51 per cent) died of heart disease in an average of ten months following examination (Table 6). Fifty-four patients with coronary disease and auricular

TABLE 5
MORTALITY IN CASES OF AURICULAR FIBRILLATION AND AORTIC INSUFFICIENCY

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
20-29	2	2		1	50	10	1		
40-49	3	1	2					3	100
50-59	3	3		1	33	9		2	67
60-69	2	2		2	100	1			
Total and average.	10	8	2	4	40	7	1	5	50

TABLE 6
MORTALITY IN CASES OF AURICULAR FIBRILLATION AND HYPERTENSIVE HEART DISEASE

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
30-39	3	2	1	1	33	1		2	67
40-49	11	5	6	7	64	5		4	36
50-59	25	16	9	12	48	11		13	52
60-69	28	16	12	14	50	12	3	11	39
70-79	2	1	1	1	50	12		1	50
Total and average.	69	40	29	35	51	10	3	31	45

TABLE 7
MORTALITY IN CASES OF AURICULAR FIBRILLATION AND CORONARY DISEASE

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
40-49	10	9	1	2	20	10	1	7	70
50-59	25	20	5	10	40	11		15	60
60-69	9	4	5	3	33	15		6	67
70-79	10	10		3	30	5	4	3	30
Total and average.	54	43	11	18	33	11	5	31	57

fibrillation were traced; eighteen (33 per cent) were found to have died of heart disease within an average of eleven months (Table 7).

It is interesting to note the lower averages of mortality in the groups made up of patients with goiter. Probably this lower mortality is owing to the fact that the disturbance which occurs, at least in the majority of instances, is not progressive after the hyperthyroidism has been relieved. It has been fairly definitely shown that the degree of cardiac insufficiency which attends both exophthalmic goiter and hyperfunctioning adenomatous goiter is not dependent so much on the intensity of the hyperthyroidism as on its duration.

Table 8 gives the data in 101 cases of exophthalmic goiter with auricular fibrillation: twenty-seven patients (28 per cent) died of heart disease within an average of twenty-

TABLE 8
MORTALITY IN CASES OF AURICULAR FIBRILLATION AND EXOPHTHALMIC GOITER

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
20-29	12	6	6	4	33	7	2	6	50
30-39	25	9	16	11	44	11	2	12	48
40-49	35	12	23	9	26	7	2	24	69
50-59	26	11	15	3	12	9	2	21	81
60-69	3	3					2	1	33
Total and average.	101	41	60	27	28	9	10	64	63

eight months following observation. The data concerning hyperfunctioning adenomatous goiter are similar: sixty patients have been traced and eighteen deaths (30 per cent) from heart disease occurred within an average of ten months following examination (Table 9).

TABLE 9
MORTALITY IN CASES OF AURICULAR FIBRILLATION AND HYPERFUNCTIONING ADENOMATOUS GOITER

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
30-39	2		2					2	100
40-49	16	4	12	2	13	8		14	87
50-59	33	6	27	11	33	10		22	67
60-69	8	3	5	4	50	13		4	50
70-79	1		1	1	100	1			
Total and average.	60	13	47	18	30	10		42	70

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Chapter VII

VENTRICULAR FIBRILLATION

The ventricles may become the seat of fibrillation in much the same manner as that in which the auricles become disturbed. Electrocardiograms of ventricular fibrillation in man are relatively few, as the disturbance, unless its duration is very brief, is incompatible with life. A very few instances of this disorder have been recorded in which there has been temporary restoration of cardiac activity. Ventricular fibrillation has been produced in animals by faradization of the ventricles, by chloroform anesthesia, and by ligation of the coronary arteries. It is undoubtedly the terminal mechanism in many

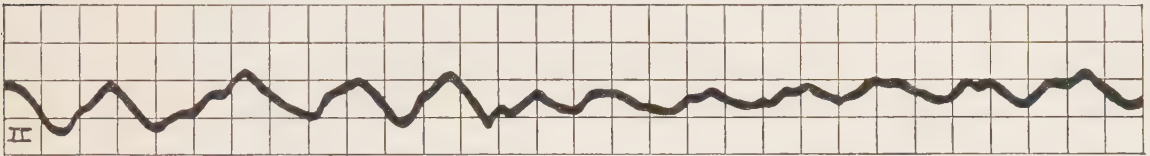


FIG. 116.—Record in lead II showing ventricular fibrillation. Record obtained, just before death, from a man aged fifty-eight years.

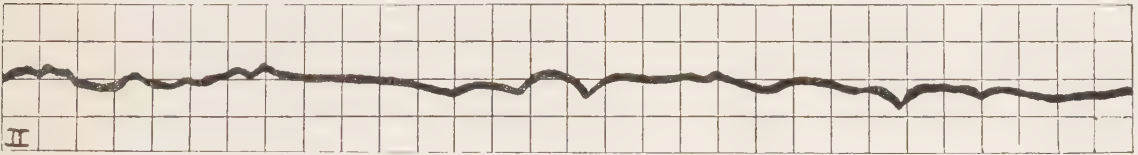


FIG. 117.—Record in lead II showing ventricular fibrillation. Note the irregular, incoördinate undulations. Record obtained just before death, from a woman, aged forty-eight years, who had chronic endocarditis with mitral stenosis.**

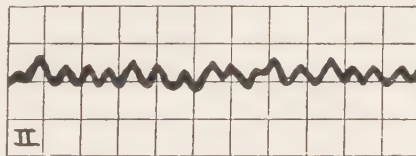


FIG. 118.—Record in lead II showing ventricular fibrillation. Note the irregular, poorly defined undulations. Record obtained from a dog just before death.**

dying hearts although exceptions to this have been noted (Figs. 337 to 368). Its presence at the time of death is not limited, by any means, to hearts invaded by disease as it clearly has been shown to exist in cases in which the heart has been entirely uninvolved. The fact that dynamic contraction of the ventricles has ceased in ventricular fibrillation renders the condition incompatible with life.

The electrocardiograms of ventricular fibrillation vary, although the general characteristics are similar. The graphs display incoördinate undulations. At times these undulations are quite well marked, but are irregular and vary greatly in amplitude as

shown in Figures 116 and 331 to 336. The undulations may be of extremely irregular low amplitude, as in Figure 117. In Figure 118 is an example of ventricular fibrillation, occurring in a dog, with extremely rapid and fairly uniform undulations.

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Chapter VIII

COMPLETE AND PARTIAL HEART-BLOCK AND DELAYED AURICULO-VENTRICULAR CONDUCTION

Heart-block results from interference with conduction of the impulse from auricles to ventricles through the auriculoventricular (His) bundle. Three types of heart-block exist: (1) When there is complete failure in conduction of impulses through the bundle, complete heart-block (dissociation) is present; (2) owing to the intermittent failure of the bundle to conduct impulses, the ventricles may fail to respond to each second, third, or fourth impulse, and then partial heart-block is said to exist, and (3) there may be only a delay in the transmission of the impulse and each auricular systole may be followed by a ventricular systole; this condition is known as delayed auriculoventricular conduction.

COMPLETE HEART-BLOCK

Complete heart-block is characterized by an extremely slow ventricular rate, usually in the vicinity of 30 beats each minute. At times, however, the rate may be considerably

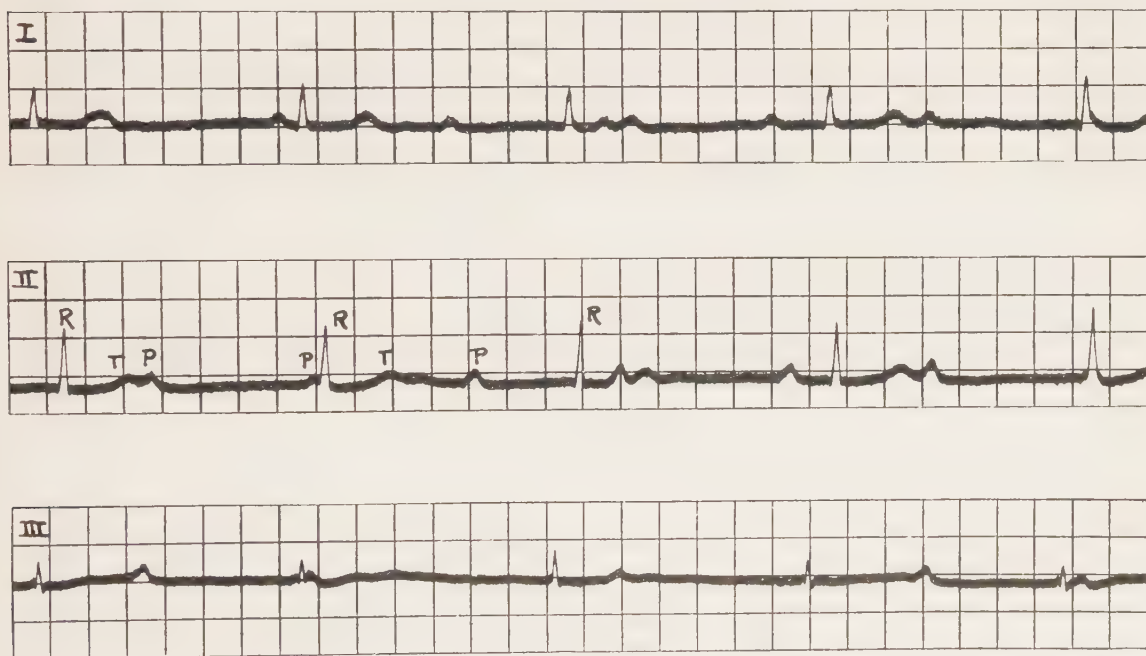


FIG. 119.—Record showing complete heart-block with complete auriculoventricular dissociation. The ventricular rate is 43, and the auricular rate is 72 contractions each minute. Record obtained from a woman, aged twenty-nine years.

higher. The ventricles are activated by a focus of irritability which becomes established in some portion of their musculature and by means of which is set up a ventricular rhythm

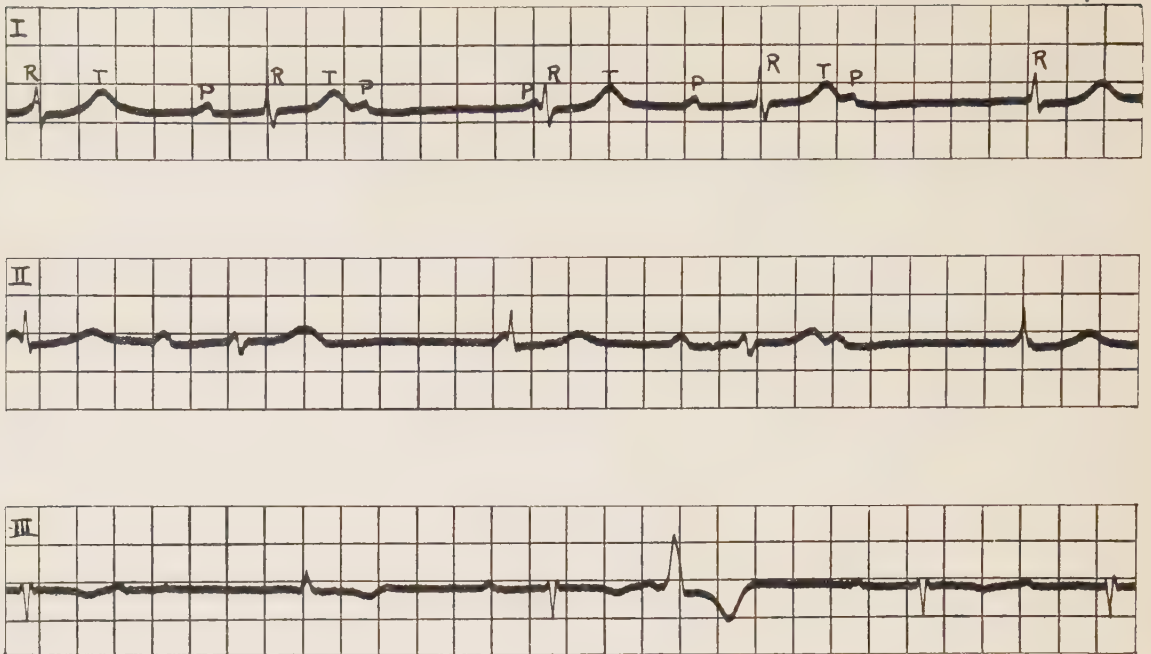


FIG. 120.—Record showing complete heart-block; the ventricular rate is 45, and the auricular rate 66 contractions each minute. Note the complete dissociation of auricles and ventricles and the variation in character of the QRS complexes. Record obtained from a woman, aged fifty-eight years, with hypertensive and coronary heart disease.

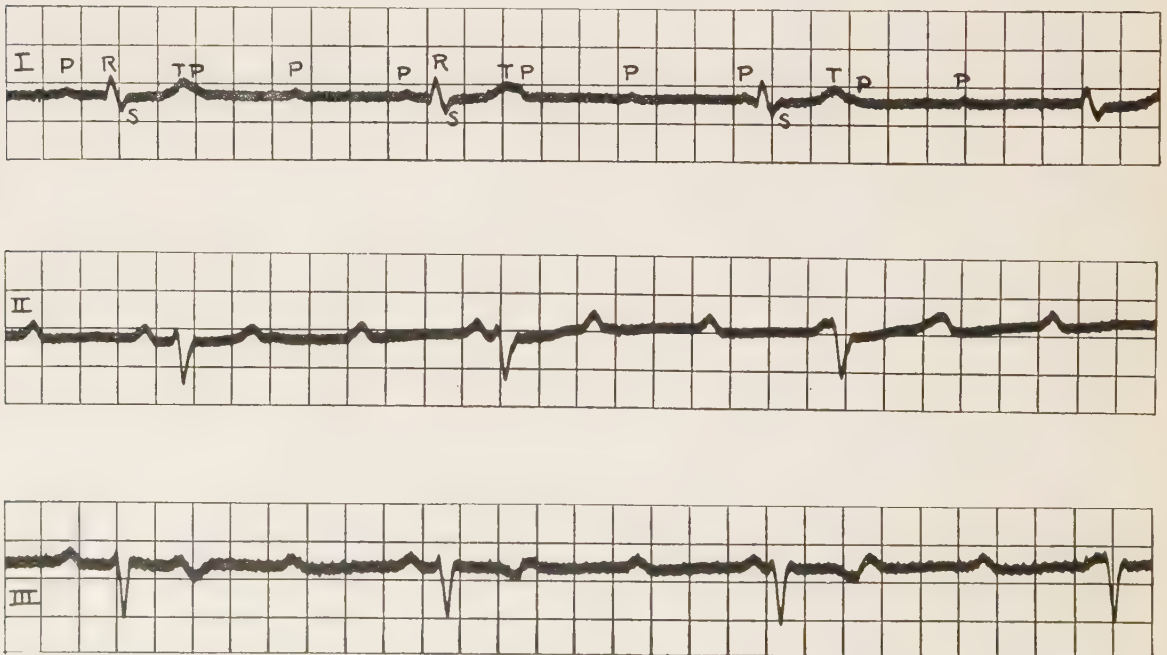


FIG. 121.—Record showing complete heart-block; the ventricular rate is 36, and the auricular rate is 100 contractions each minute. The P waves continually bear different relationships to the QRS complexes, although the P wave sequences are very regular. Slight aberration of the QRS complexes occurs, and there is preponderance of the left ventricle. Record obtained from a man, aged seventy-two years, with coronary sclerosis.

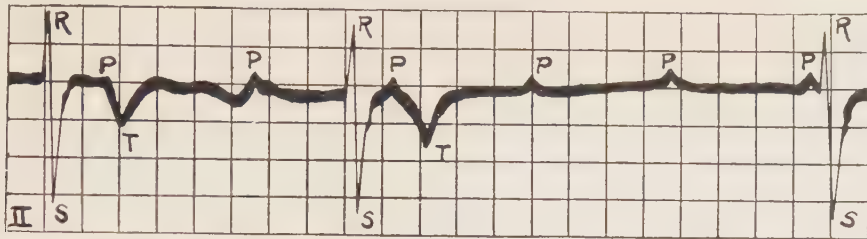


FIG. 122.—Record in lead II showing complete heart-block. Note the deep inversion of the T waves. Record obtained from a man, aged sixty-two years, with coronary and hypertensive heart disease.**

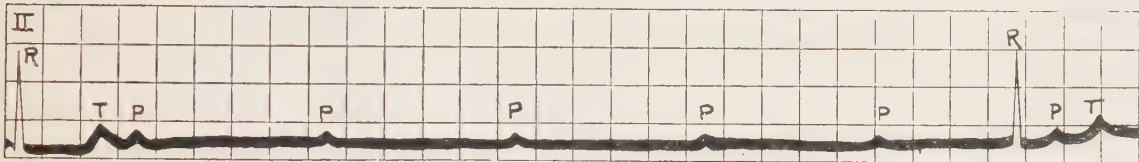
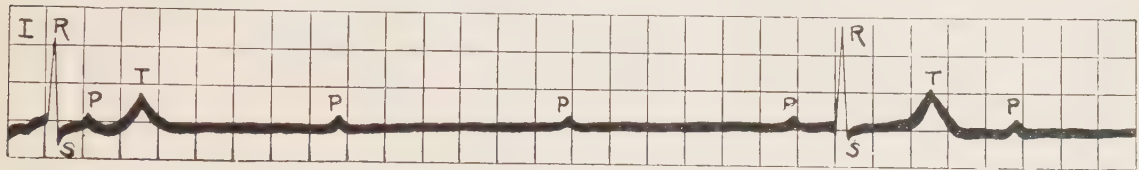


FIG. 123.—Records in leads I and II showing complete heart-block, with long periods of ventricular asystole during which convulsive syncope occurred. Record obtained from a man aged forty-two years.**

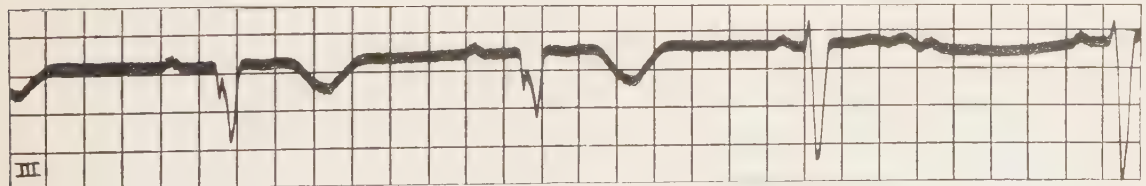
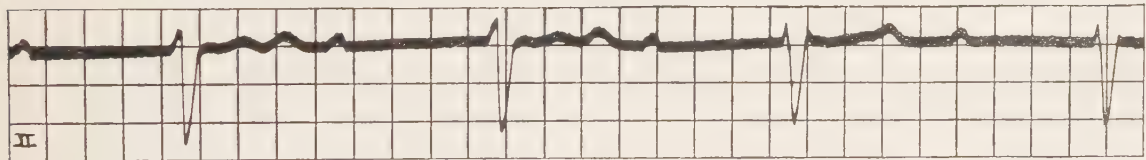
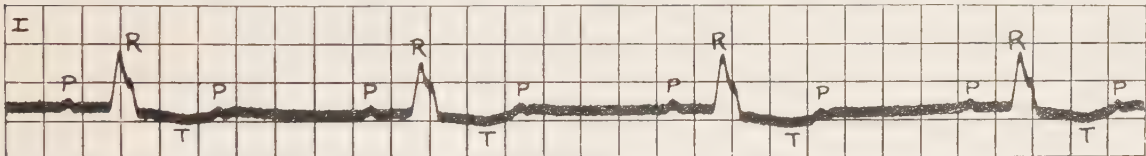


FIG. 124.—Record showing temporary complete heart-block. The total auriculoventricular dissociation is apparent. The ventricular rate is 37, and the auricular rate 75 contractions each minute. This record, and the subsequent one, were obtained from a man, aged forty-five years, with coronary sclerosis accompanied by attacks of convulsive syncope.

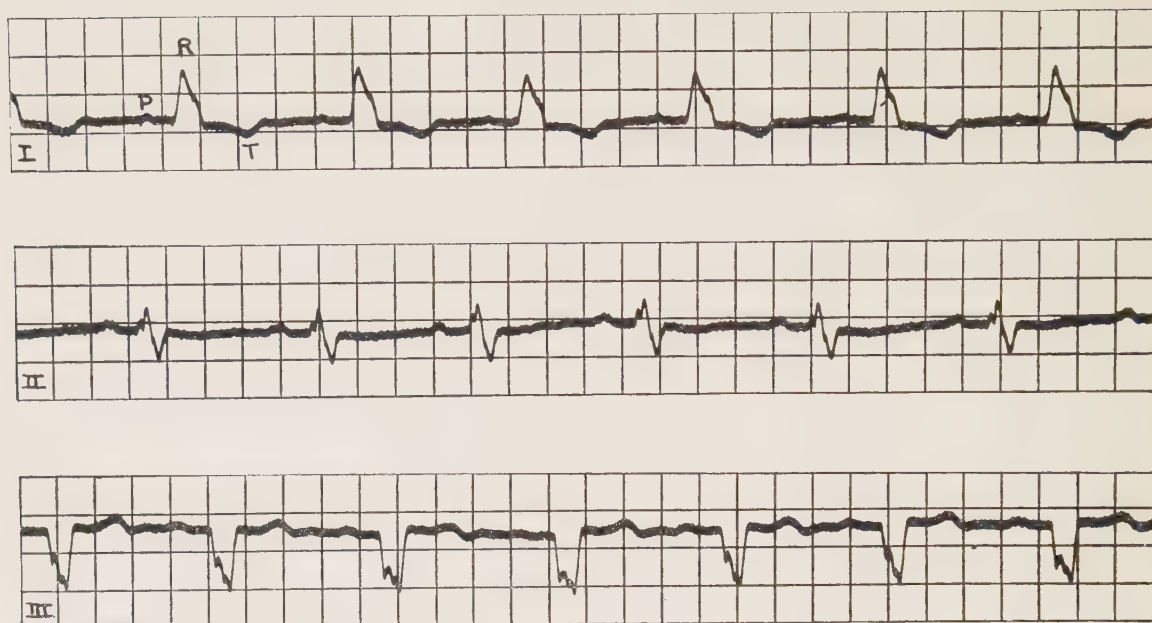


FIG. 125.—Record of the same patient who is represented in Figure 124, taken eight months later, showing the abolishment of complete heart-block and the presence of complete right bundle-branch block. The QRS complexes in all leads are deformed and the base width of the complexes is 0.14 second. The heart rate is 66 contractions each minute. Note the inversion of the T components in lead I.

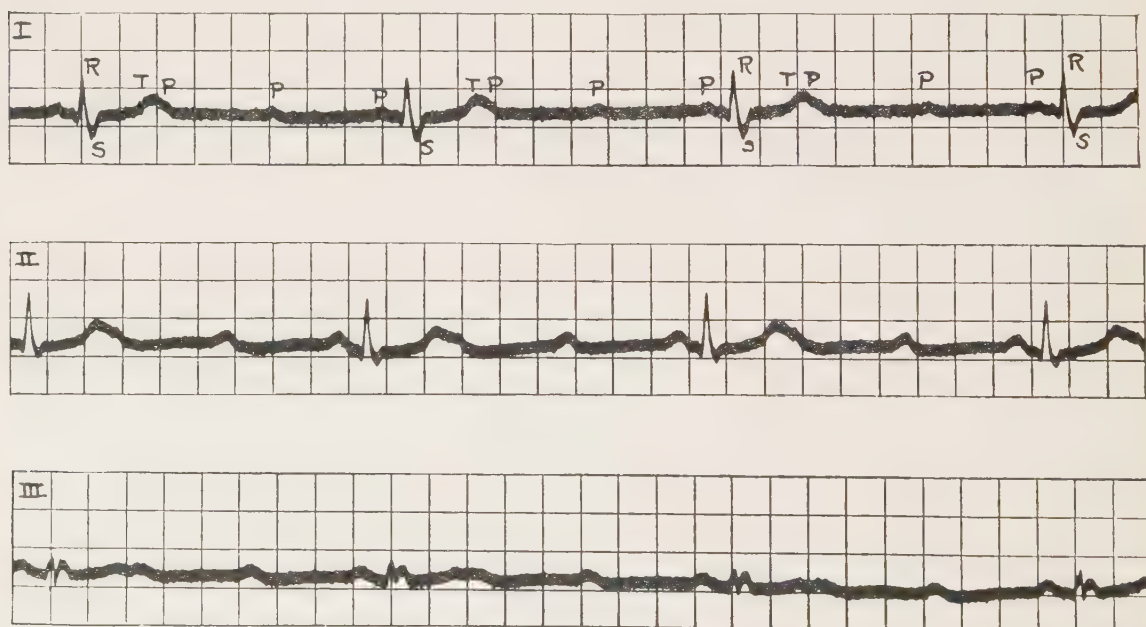


FIG. 126.—Record in all leads showing 3 : 1 partial heart-block. The QRS complexes are quite normal in leads I and II, but are notched and of very low amplitude in lead III. Three P waves, regularly placed, occur in every cycle. The records in this figure, and those in the succeeding Figures 127 to 134, were obtained from a man, aged seventy-five years, who presented numerous interesting and some very unusual disturbances in auriculoventricular conduction. Numerous attacks of convulsive syncope occurred during the period of observation. Necropsy revealed the presence of a calcified nodule about 1 by 1 cm. encroaching on the auriculoventricular bundle. Considerable fibrosis of the bundle had occurred.

plexes that are normal in contour (Figs. 119, 120, 123, 135, and 136). At times, however, there is definite deformity of the QRS complexes which consists in notching of the

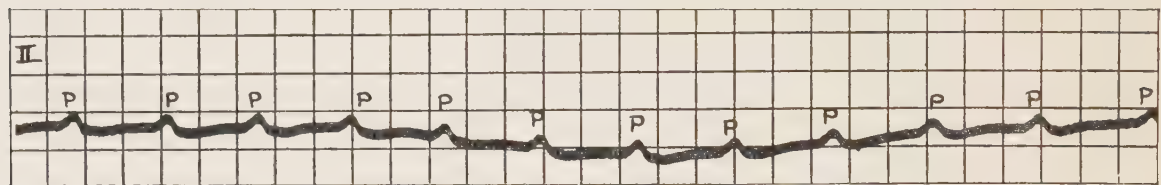
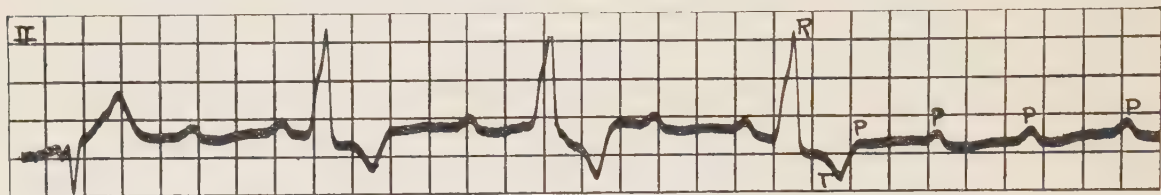
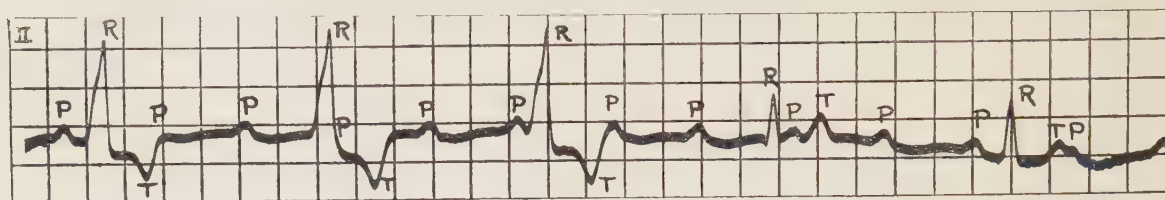


FIG. 129.—Continuous record in lead II of the same patient (Fig. 128). It shows varying degrees of partial block with many bizarre QRS complexes of high amplitude, indicating the temporary establishment of idioventricular rhythm. An occasional supraventricular type of ventricular response is seen. In the latter part of the second strip ventricular asystole occurs, and in Figure 130 a block of 24 : 1 is observed. During this period of ventricular asystole, which lasted 15.6 seconds, convulsive syncope occurred.

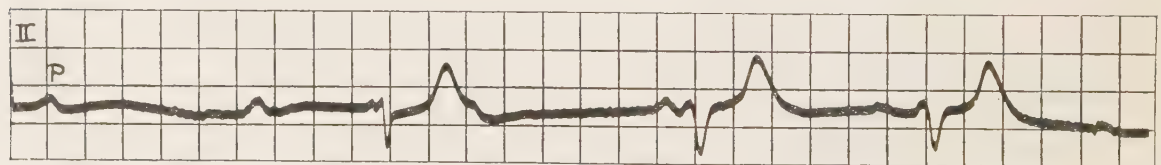
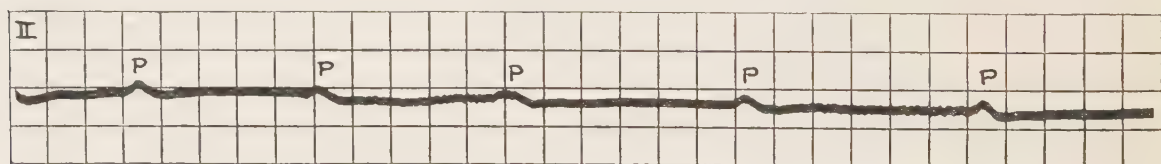


FIG. 130.—Continuous record of the same patient (Fig. 129), in lead II, showing the termination of a long period of ventricular asystole. The idioventricular rhythm in the preceding figure apparently had its origin in the right ventricle; the rhythm of recovery, as seen in this figure, apparently arises in the left ventricle.

components and increase in the duration of execution, as seen in Figures 121, 124, 126, 127, and 129. These abnormal complexes may appear in single leads or in all leads. Occasionally changes in form of successive complexes occur (Fig. 124). Inversion, or

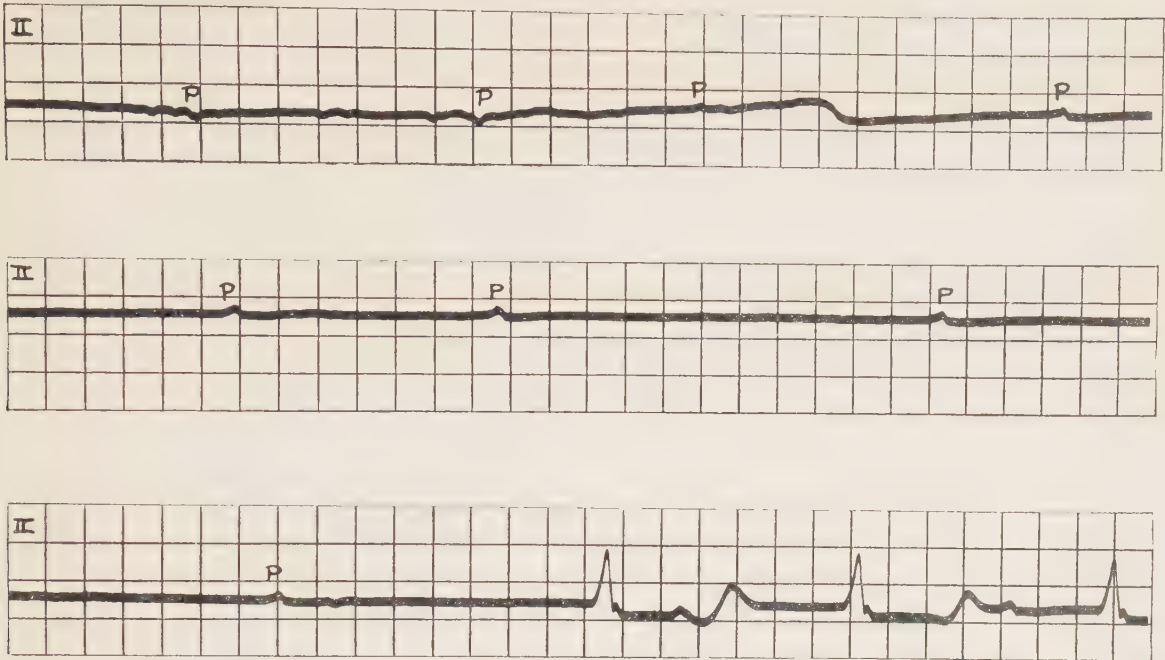


FIG. 131.—Records in lead II, continuous with those in Figure 130, again show a long period of ventricular asystole occurring with convulsive syncope. Note the marked irregularity of the P waves and the long distances between them.

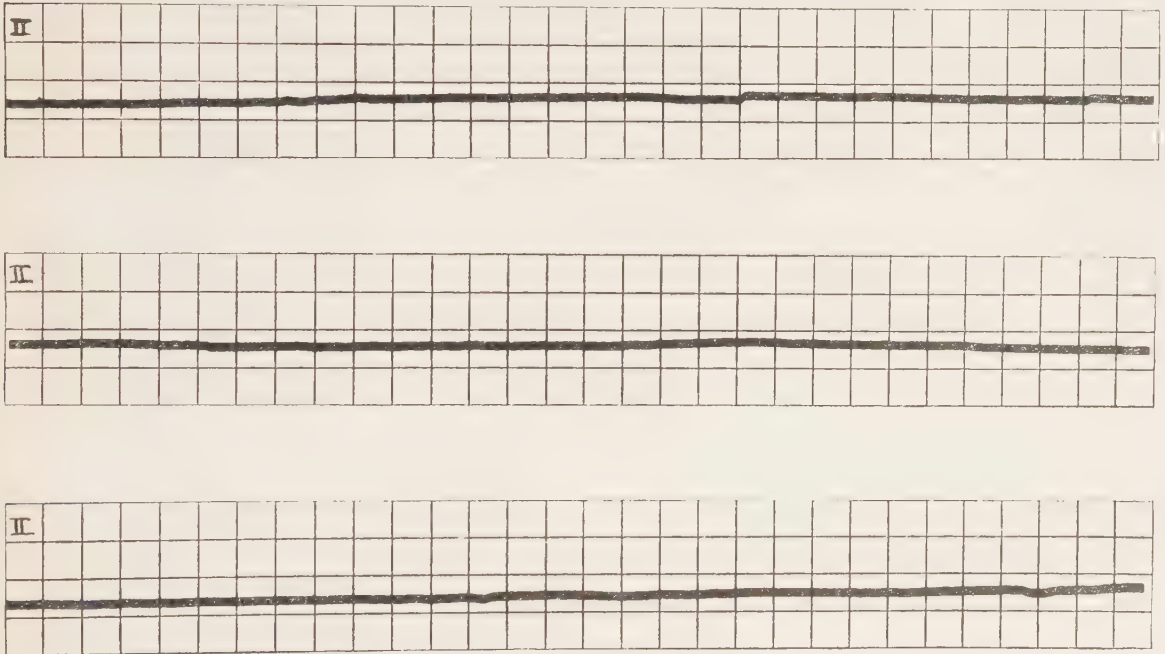


FIG. 132.—Records in lead II, continuous with those in Figure 131 and with those in the succeeding figure, show a remarkably long period of complete cardiac asystole, which lasted about 24.2 seconds. The occasional irregular undulations of the record are probably artifacts. The patient appeared to be dead at this time; he recovered for about forty-two hours.

negativity, of the T component is not unusual in isolated or combined leads (Figs. 120, 122, 124, and 129). Interruption of the regular, idioventricular rhythm by ventricular

premature contractions is at times observed (Fig. 120). The auricular P waves occur in varying relations to the ventricular waves owing to the independent rhythm and rate of the latter. The auricular cycle, or P-P interval, is usually regular and its relationship to the other waves is constantly changing. This is clearly shown in Figure 119.

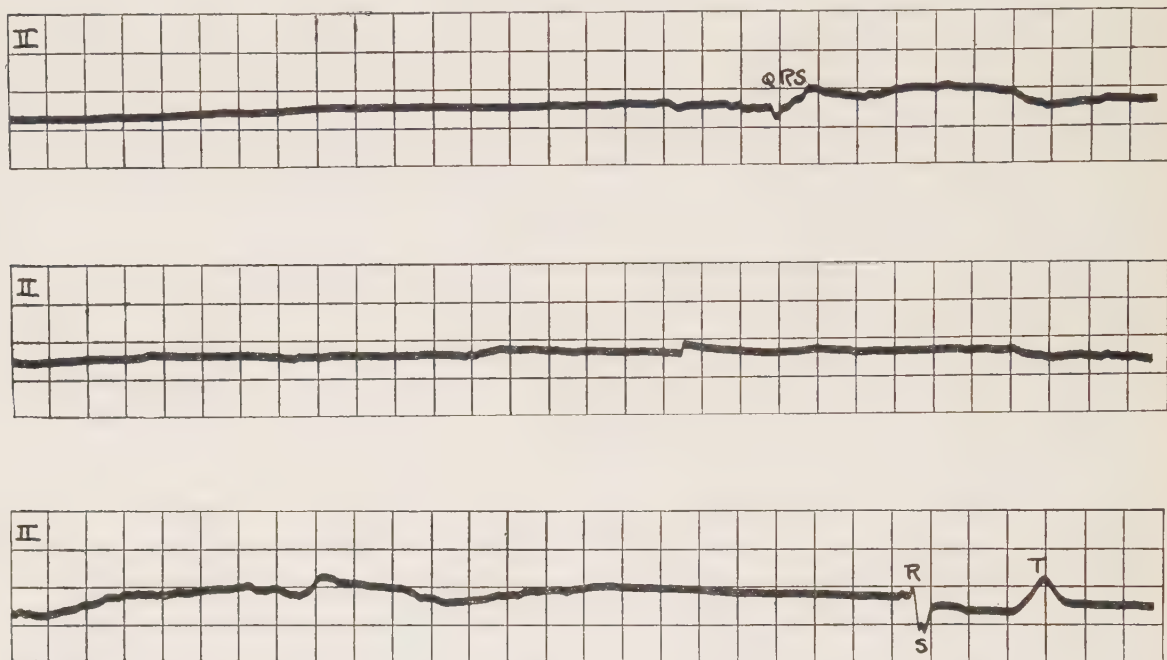


FIG. 133.—Records in lead II continuous with those in Figure 132. In the latter portion of the first strip an atypical ventricular complex of low amplitude occurs. A more distinct complex appears near the end of the last strip.

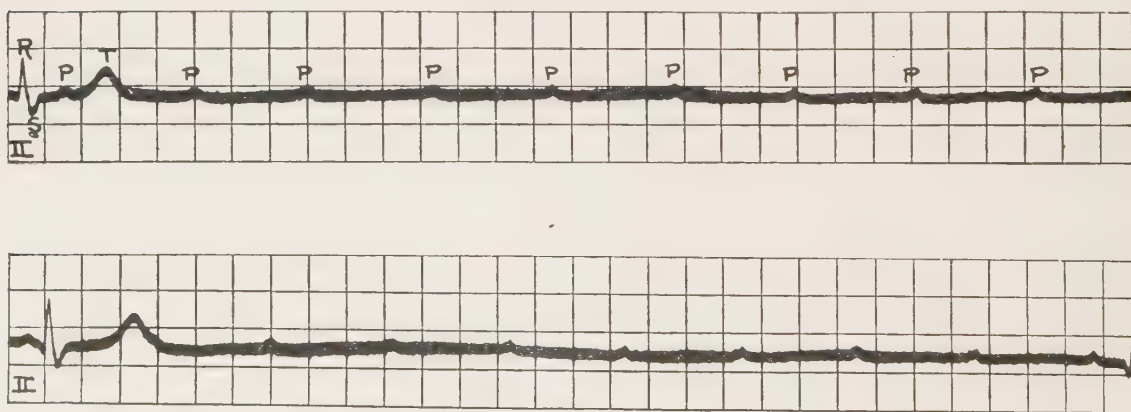


FIG. 134.—Records of the same patient (Fig. 133) in lead II, showing other examples of high-grade block occurring in the same patient.

Occasionally auricular fibrillation is associated with established complete heart-block.

At times, and invariably with convulsive syncope, long periods of ventricular asystole occur. In these tracings the degree of block may vary from 4 : 1 or 5 : 1 (Fig. 123) to

24 : 1 (Figs. 129 and 130). At other times unusually long periods of complete cardiac asystole may occur (Figs. 132 and 133).

Complete heart-block may be interchangeable at times with partial block and with normal sinus rhythm. Such events are clearly recorded in Figures 126 to 134. These

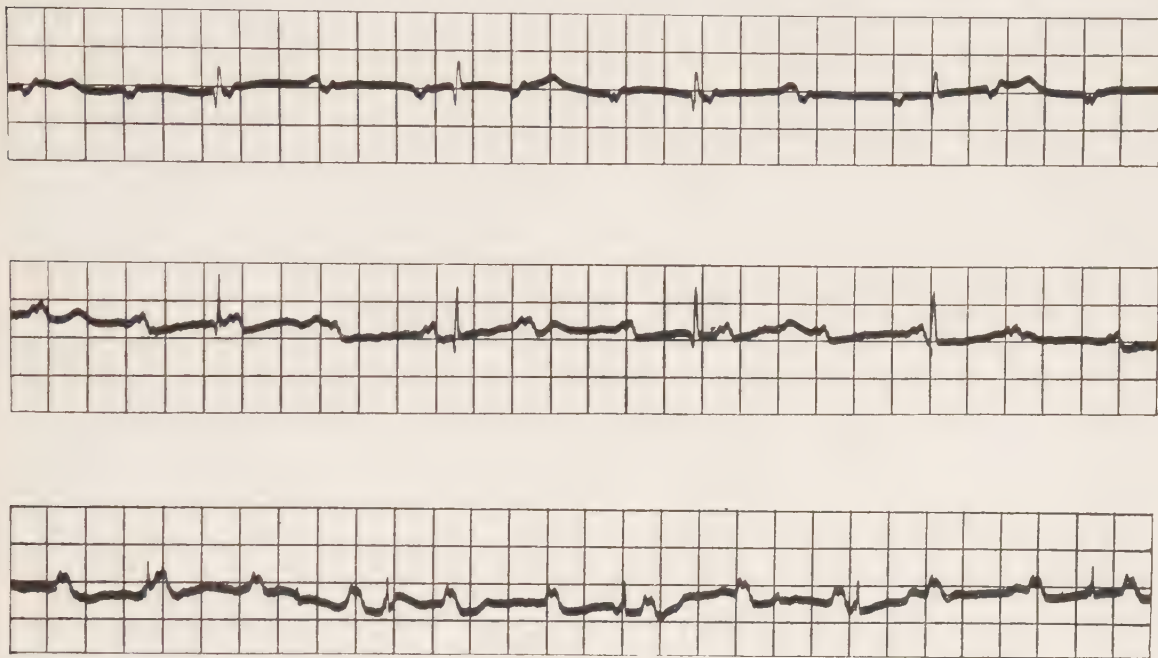


FIG. 135.—Record showing complete heart-block; the ventricular rate is 47, and the auricular rate 116 contractions each minute. Note the notching of the P waves in all leads and the inversion of the P waves in lead I. Record obtained from an infant, aged six days, with congenital heart disease. There was complete situs transversus of all the abdominal and thoracic viscera except the ventricles of the heart.

electrocardiograms were all obtained during a period of a few days. In Figure 126, 3 : 1 partial heart-block is represented; and this is followed, in Figure 127, by 2 : 1 partial heart-block. In the succeeding record, Figure 128, normal sinus rhythm was estab-

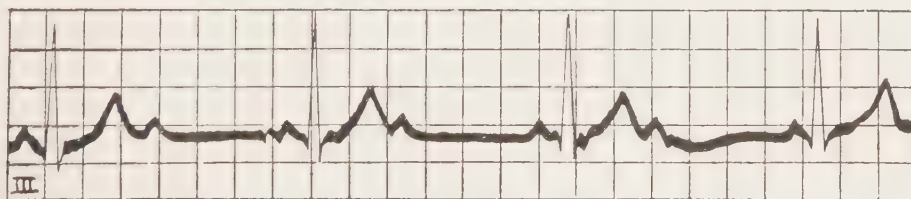


FIG. 136.—Record in lead III showing 2 : 1 partial heart-block. Note the high amplitude of the T waves. Record obtained from a man, aged fifty-seven years, with coronary and hypertensive heart disease accompanied by attacks of convulsive syncope.**

lished. In Figures 129 and 130 the record of complete heart-block is followed by that of a long period of ventricular asystole and the subsequent resumption of complete heart-block. Figures 132 and 133 represent long periods of complete cardiac asystole with, finally, temporary cardiac recovery. Figures 124 and 125 also illustrate the temporary

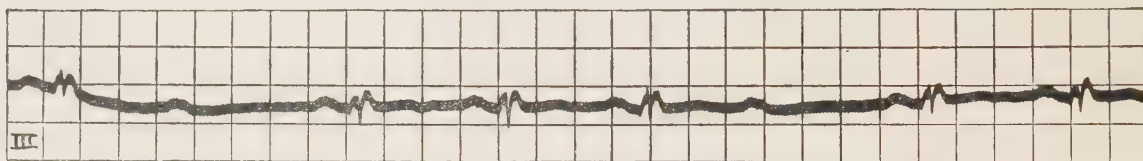
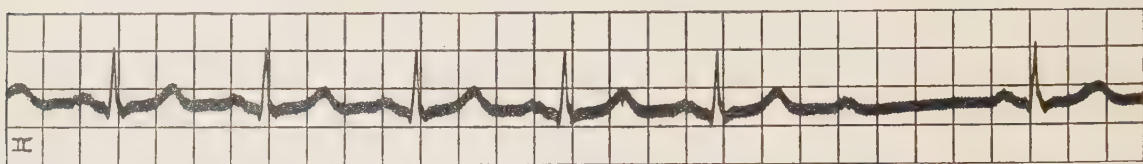
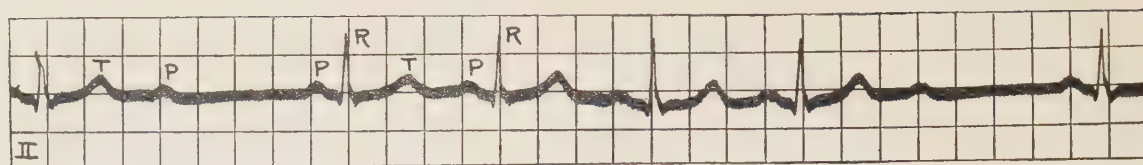


FIG. 137.—The upper two strips, in lead II, and the lower strip, in lead III, show intermittent 2 : 1 heart-block. Such transient partial heart-block is unusual. Record obtained from a man, aged seventy-five years, with attacks of convulsive syncope.

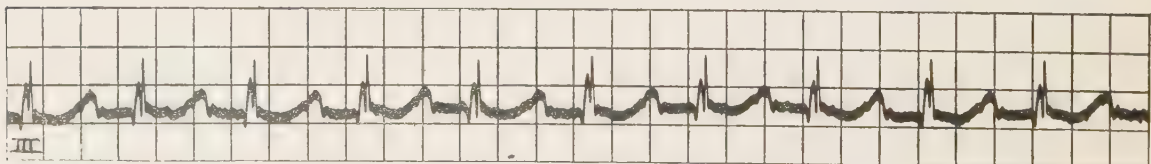
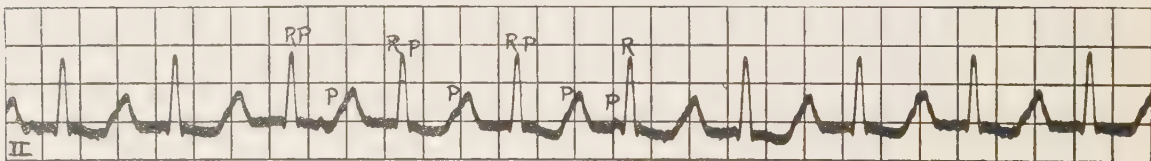
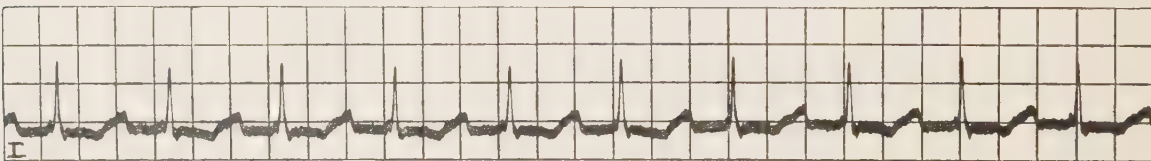


FIG. 138.—Record showing complete dissociation of auricles and ventricles with rapid ventricular rate—100 contractions each minute. Note the general tendency to the occurrence of 2 : 1 ratio. The T waves in lead I are inverted and the QRS complexes in lead III are notched. Record obtained from a man, aged thirty-six years, who had chronic rheumatic endocarditis with aortic insufficiency accompanied by marked cardiac hypertrophy.

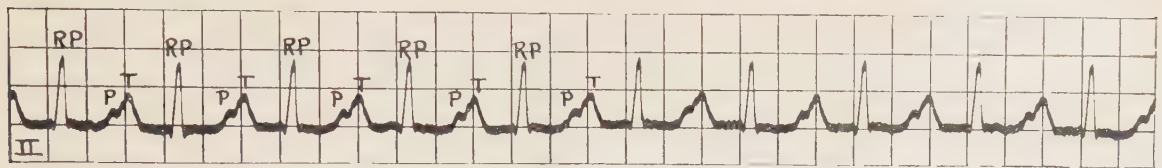


FIG. 139.—Record of the same patient (Fig. 138) taken in lead II, showing 2 : 1 heart-block. The T waves are constantly and regularly deformed by the coincidental occurrence of P waves.

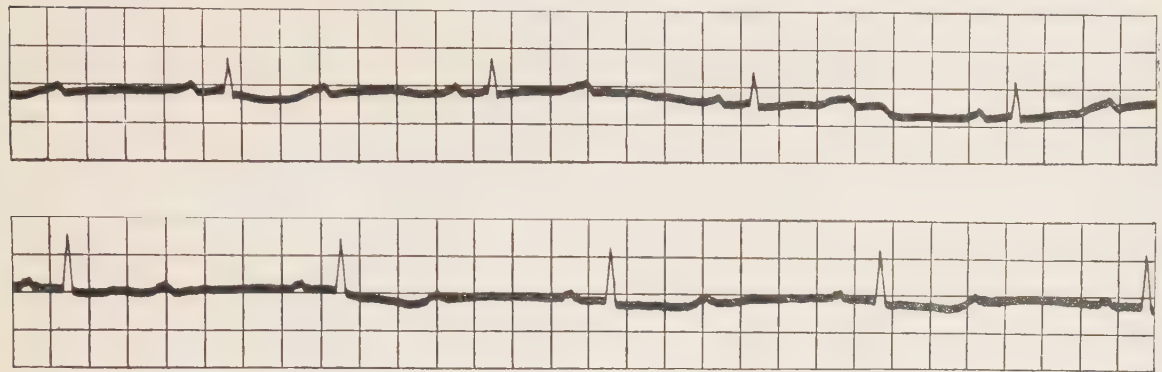


FIG. 140.—Record showing 2 : 1 partial heart-block in a man, aged fifty-eight years, with coronary and hypertensive heart disease.

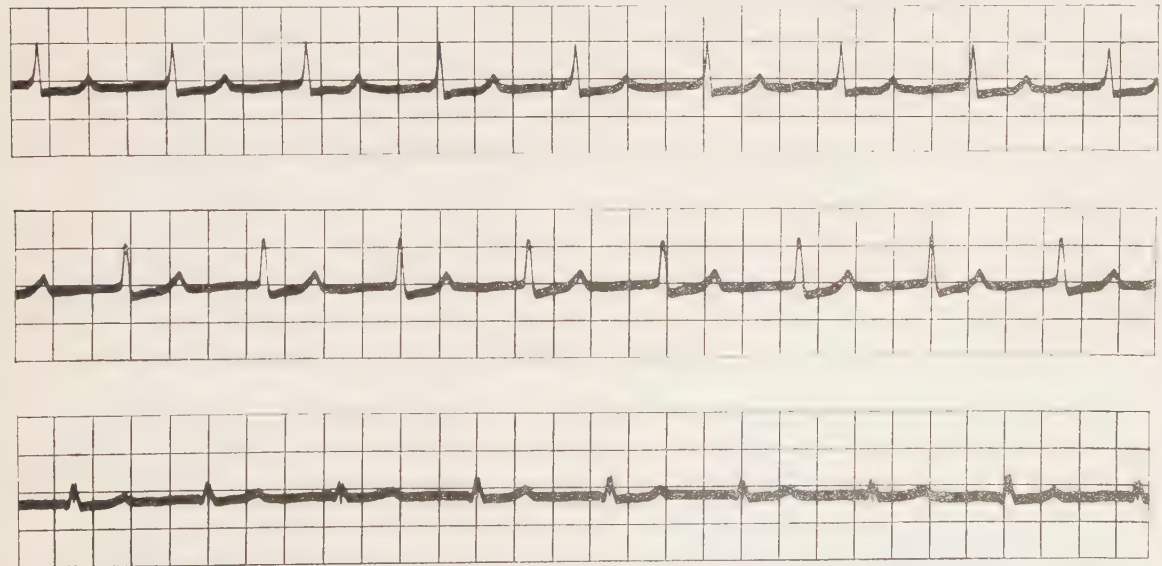


FIG. 141.—Record from the same patient (Fig. 140) obtained two days later and showing nodal rhythm, the inception of which occurred spontaneously.

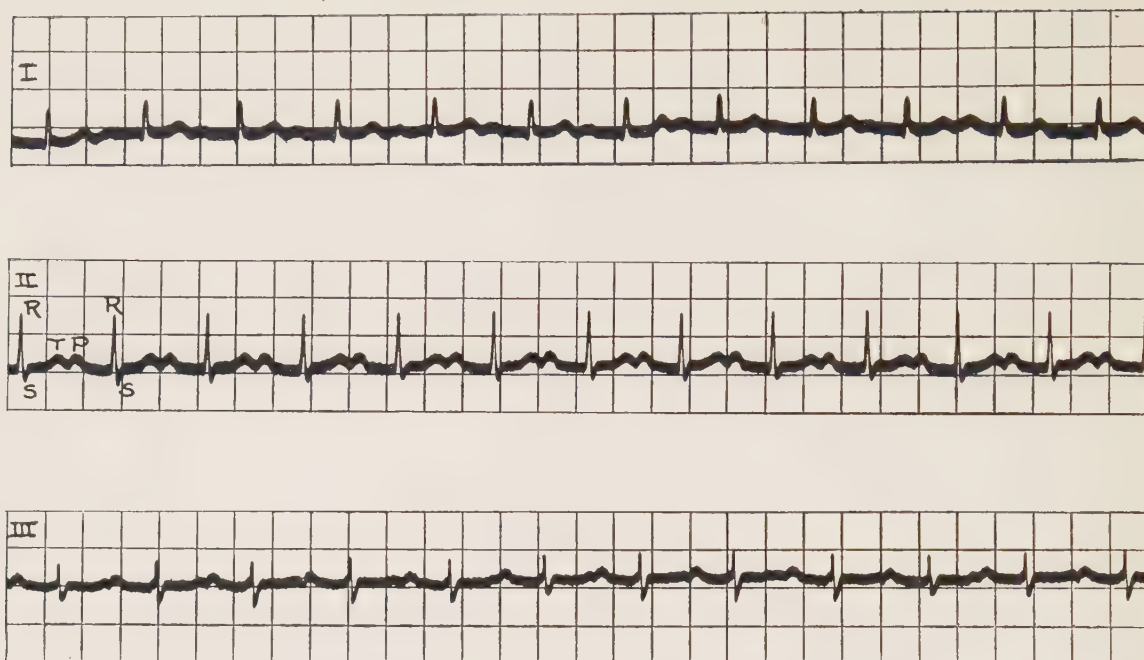


FIG. 142.—Record showing delayed auriculoventricular conduction; the P-R interval is 0.24 second. Slight preponderance of the left ventricle is evident. Record obtained from a woman, aged twenty-six years, with exophthalmic goiter.

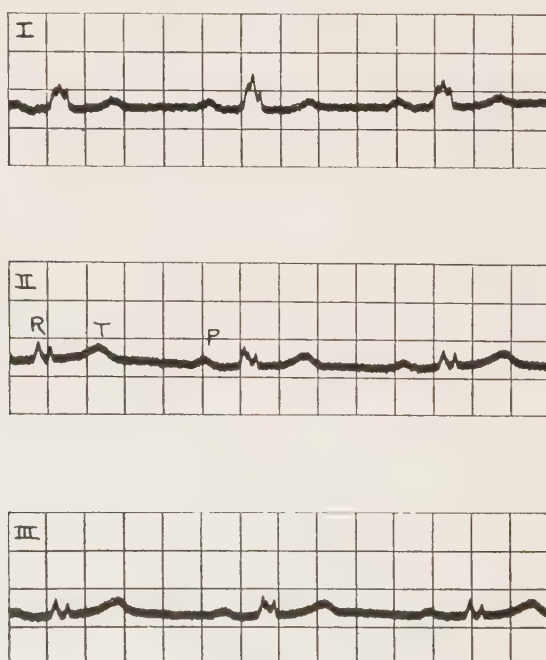


FIG. 143.—Record showing delayed auriculoventricular conduction and notching of the QRS complexes in all leads. The P-R intervals are 0.24 second. Record obtained from a man, aged seventy-four years, with coronary sclerosis.

presence of complete heart-block; the latter record, made eight months later, shows sinus rhythm associated with aberrant QRS complexes in all leads.

Occasionally, in cases in which demonstrable evidence of organic heart disease does not exist, complete auriculoventricular block occurs for a short period; then it spontaneously disappears and normal sinus rhythm returns. Similar disappearance of block

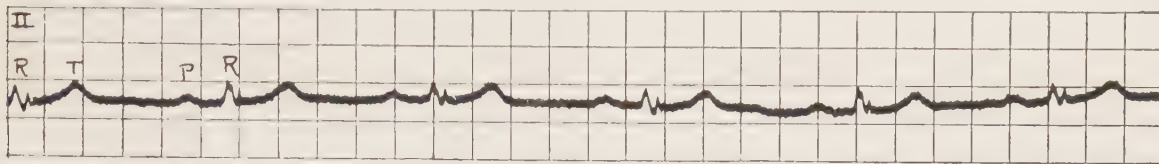


FIG. 144.—Record in lead II showing delayed auriculoventricular conduction; the P-R interval is 0.24 second. There is notching of the QRS complexes. Record obtained from a man, aged seventy-five years, with coronary sclerosis.

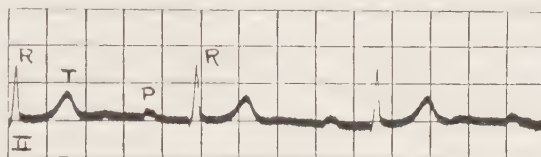


FIG. 145.—Record in lead II showing delayed auriculoventricular conduction. The P-R interval is 0.25 second. Record obtained from a man, aged sixty-three years, with cardiac hypertrophy and aortic sclerosis.

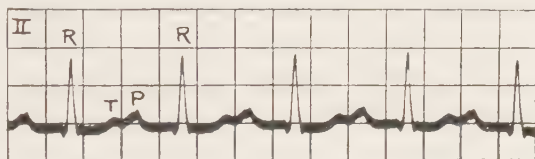


FIG. 146.—Record in lead II showing delayed auriculoventricular conduction; the P-R interval is 0.26 second. Record obtained from a man, aged fifty-seven years, with coronary sclerosis.

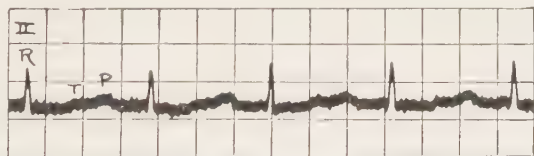


FIG. 147.—Record in lead II showing delayed auriculoventricular conduction. The P-R interval is 0.28 second. Record from a man, aged seventy-four years, with coronary sclerosis.

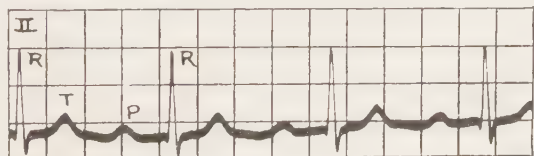


FIG. 148.—Record in lead II showing delayed auriculoventricular conduction; the P-R interval is 0.28 second. Record obtained from a man, aged thirty-four years, who had chronic rheumatic endocarditis with aortic insufficiency.

has been observed following the subcutaneous administration of atropin and, under these conditions, it is presumptive evidence that the block was related to vagal influences.

Complete heart-block in man results from various causes: chiefly from lesions of the auriculoventricular bundle or node; from asphyxia; from drugs, such as digitalis, strophan-

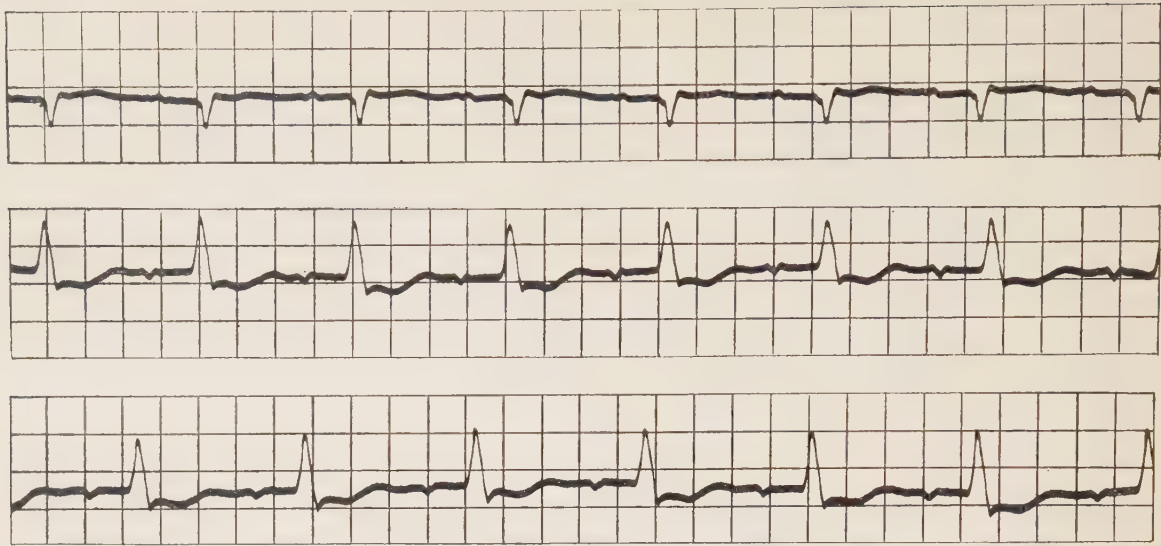


FIG. 149.—Record showing delayed auriculoventricular conduction; the P-R interval is 0.25 second. Note the inversion of the P waves in leads II and III. Record obtained from a man, aged fifty-nine years, with hypertensive heart disease.

thus, and squill, and occasionally from vagal influences. Apparently the most common lesion involving the junctional conducting system results from atherosclerosis. The coronary branches which supply the auriculoventricular node and bundle are probably the seat of obliterative disease in many cases, and this interferes definitely with the conduction of impulses even though a gross lesion of the conduction paths is not apparent. This apparent discrepancy of cause and effect has been the basis of considerable controversy in cases in which the bundle has been examined microscopically.

Other lesions which are responsible for complete heart-block are gummas, areas of calcification, infarction of the septum, ulcerative endocarditis, and various types of cellular infiltration and degeneration.

TABLE 10
MORTALITY IN CASES OF COMPLETE HEART-BLOCK

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
20-29	4	3	1	1	25	14	1	2	50
30-39	6	5	1	3	50	21		3	50
40-49	13	7	6	9	69	9	1	3	23
50-59	11	7	4	7	64	11		4	36
60-69	23	19	4	17	75	10		6	26
70-79	3	3		2	67	5		1	33
Total and average.	60	44	16	39	65	11	2	19	32

Complete heart-block is not incompatible with reasonably good health although the majority of the patients manifest significant symptoms and are in constant danger, especially when convulsive syncope occurs.

In general, the prognosis in complete heart-block is grave, especially when convulsive syncope exists. The cardiac mortality in sixty cases is shown in Table 10; thirty-nine patients (65 per cent) died in an average of eleven months following examination.

PARTIAL HEART-BLOCK

Partial heart-block differs from complete block in that some auricular impulses are conducted through the junctional tissues to provoke ventricular response. Partial block, with 2 : 1 relationship, is the type most frequently observed; rates of 3 : 1, 4 : 1, and so forth, are less common (Figs. 126, 127, 129, 136, 137, 139, and 140). Partial block may exist as a temporary mechanism (Fig. 137) and is often associated with complete block. Probably it is rather frequently the forerunner of established complete block.

Occasionally records of partial block, attended by relatively rapid ventricular rates, are observed. Records of this type often are difficult to interpret. An example of this type is seen in Figures 138 and 139, in which the ventricular rate is 100 and the general features of the electrocardiogram are unusual. With careful study, however, it is seen that a general tendency to a 2 : 1 auriculoventricular ratio exists. In Figures 140 and 141 it is interesting to note the establishment of nodal rhythm following a period of partial block.

The causes of partial block are also those of complete block, and the clinical importance of both conditions is similar.

DELAYED AURICULOVENTRICULAR CONDUCTION

Delayed conduction between auricles and ventricles may result from disease of the auriculoventricular bundle or node, from asphyxia, from certain drugs, and from vagal influences.

The degree of delay in conduction is variable in different patients but is usually constant in the same patient. The electrocardiograms display a P-R interval longer than the normal interval of from 0.13 to 0.21 second. Prolongation of the P-R interval from 0.24 to 0.32 second is not unusual, and prolongations much greater than these have been observed (Figs. 142 to 149). Associated graphic abnormalities are often observed, such as aberration of the QRS complexes (Figs. 143 and 144) or inversion (negativity) of the T waves. Occasionally inversion of the P waves occurs in isolated or combined leads, as shown in Figure 149.

Delayed auriculoventricular conduction occurs under the same pathologic conditions that result in complete and partial heart-block. I have observed a number of cases in which, over a period of several years, the transitional stages from delayed auriculoventricular conduction to complete heart-block were disclosed. During recent years the occurrence of delayed auriculoventricular conduction in acute rheumatic fever has received considerable attention. Presumably this abnormality denotes early invasion of the heart by the disease.

Considerable caution is necessary in attaching importance to the presence of prolongation of the P-R interval in the electrocardiogram. It is not an unusual finding in

routine electrocardiography and often it may be a transitory phenomenon. In at least half of the cases in which I have observed this phenomenon, the subcutaneous administration of a physiologic dose of atropin has restored the electrocardiogram to normal within a period of ten to thirty minutes. Such a release is certainly presumptive evidence of the vagal origin of many cases of delayed auriculoventricular conduction. Therefore it is unsafe to draw the definite conclusion from one record only, that organic disturbance in impulse transmission is causing the abnormality. Except when a definite contraindication exists, patients with delayed auriculoventricular conduction always should be subjected to the atropin test.

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Chapter IX

INCOMPLETE AND COMPLETE BUNDLE-BRANCH BLOCK

Much uncertainty and difference of opinion have existed regarding the meaning of changes in form and delay in execution of the QRS complexes. Investigations conducted during the last few years, however, have given a much clearer idea regarding such graphic changes.

It has been shown that the form of the QRS complex is dependent on the course of the excitation wave, through the ventricular musculature. The duration of the QRS complex depends on the time consumed by the wave of excitation in completing its course.

Lewis and his coworkers have demonstrated that the wave of excitation follows a definite route in its spread over the ventricular muscle: it enters the ventricles by way of the auriculoventricular (His) bundle, passes along the two main branches of this structure and along their ramifications, and spreads through the Purkinje plexus which lines the inner surface of both chambers. The wave of excitation then extends outward from the Purkinje tissue, through the ventricular walls, toward the outer surface of the heart. Its rate of travel is ten times more rapid in the special conducting system than in ordinary ventricular muscle.

Wedd and Stroud recalculated the original experimental curves of Lewis and introduced a correction for the full development of the spread of the wave and excitation in the ventricle. They then observed that this period of spread corresponded with the duration of the QRS complex in the electrocardiogram.

It is necessary to revert to some of the earlier experimental studies in order to visualize certain electrocardiographic abnormalities. The studies of Eppinger and Rothberger in 1910 are important. Working with dogs, they developed a technic which permitted them to sever the right or the left main branch of the auriculoventricular bundle. After the right branch was severed, the normal ventricular electrocardiogram was replaced by a large, diphasic QRS complex. In a single lead from the esophagus and rectum, the QRS complex was directed downward, its duration was increased, and the T wave was directed upward and exaggerated in amplitude. After section of the left branch, similar changes in the electrocardiogram occurred but with a reversal in the direction of the complexes.

Shortly after these observations, Eppinger and Stoerck published electrocardiograms of two cases, with necropsy data, in which lesions that completely divided the right bundle-branch were demonstrated. Large, diphasic QRS complexes occurred in all leads, with greater amplitude in leads I and III and with intervals prolonged beyond normal. The QRS complexes were directed upward in lead I and downward in leads II and III.

In 1913 Rothberger and Winterberg repeated the previous experimental work, using the three customary leads. After the division of one of the main bundle-branches, typical diphasic complexes were obtained; but the QRS complexes in leads I and III were in the same, and not in the opposite direction, as in the cases reported by Eppinger and Stoerck.

These experiments in which dogs were used were reinvestigated by Lewis in 1916. He found that in the majority of the experiments in which he severed the right bundle-

branch the QRS complexes were directed downward in all leads. He termed these records "concordant." In a few similarly treated animals, the QRS complexes were directed upward in lead I, and such records he called "discordant." There was apparent discrepancy in the observation that right bundle-branch block in human beings resulted in discordant electrocardiograms, *whereas in most dogs* it resulted in concordant records. Lewis showed that these differences were the result of anatomic variations: the hearts of animals which produced discordant electrocardiograms showed less bridging of the cavity of the left ventricle by ramifications of the left bundle-branch than did those which produced concordant curves.

It is now generally believed that the increased duration of the QRS complex is the most reliable criterion of complete bundle-branch block, although the degree of the deflec-

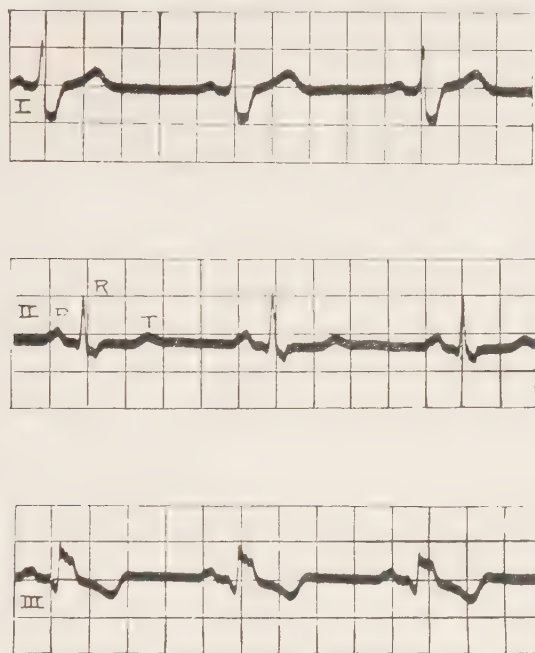


FIG. 150.—Record showing incomplete bundle-branch block of slight degree. The QRS interval in leads I and II is 0.12 second, and in lead III, 0.16 second. Note the peculiar notching and the contour of the QRS complexes in lead III. The T waves in lead III are inverted. Record from a man, aged fifty-seven years, with hypertensive heart disease.

tions of amplitude cannot be overlooked. The QRS interval usually ranges between 0.12 and 0.18 second and occasionally it is even more. Lewis had called attention to the possibility of error in placing too much reliance on the graphs when the QRS interval is 0.1 second or slightly more, as they may simulate curves of left ventricular preponderance. The prolonged QRS interval of complete bundle-branch block is believed to result chiefly from retardation of the spread of the excitation through the interventricular septum. Thereby, the length of the QRS interval is influenced by the thickness of the septum, which often is increased in greatly hypertrophied hearts.

The high amplitude of the QRS complexes is a striking feature of the records of complete bundle-branch block and results, according to Wilson and Herrmann, chiefly from lack of balance. These investigators called attention to the fact that, in normal hearts, the effects produced by the activation of the free wall of the left ventricle and those produced

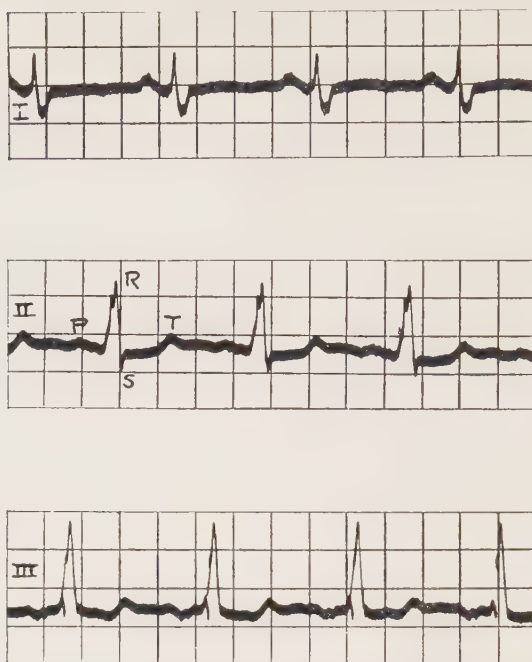


FIG. 151.—Record showing incomplete bundle-branch block. The QRS interval is 0.12 second. The notching is well marked in lead II. Record from a woman, aged twenty-eight years, who had chronic rheumatic endocarditis with mitral stenosis.

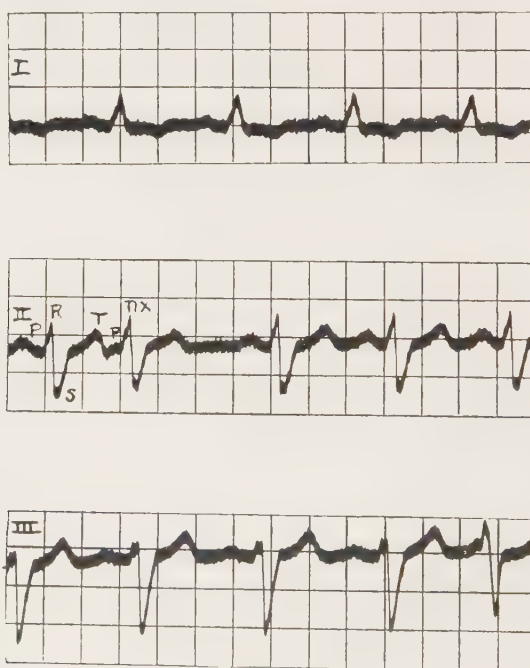


FIG. 152.—Record of incomplete bundle-branch block with inversion of T waves in lead I. A nodal (A-V) premature contraction occurs as the second complex in lead II. The QRS interval is 0.12 second. Record obtained from a man, aged sixty-nine years, with coronary sclerosis.

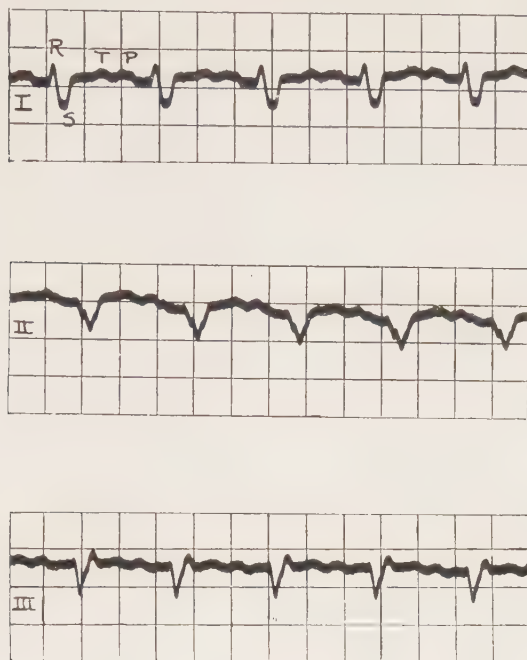


FIG. 153.—Record of incomplete bundle-branch block showing bizarre QRS complexes of low amplitude in all leads. The QRS interval is 0.12 second. Record obtained from a man, aged seventy-one years, with coronary sclerosis.

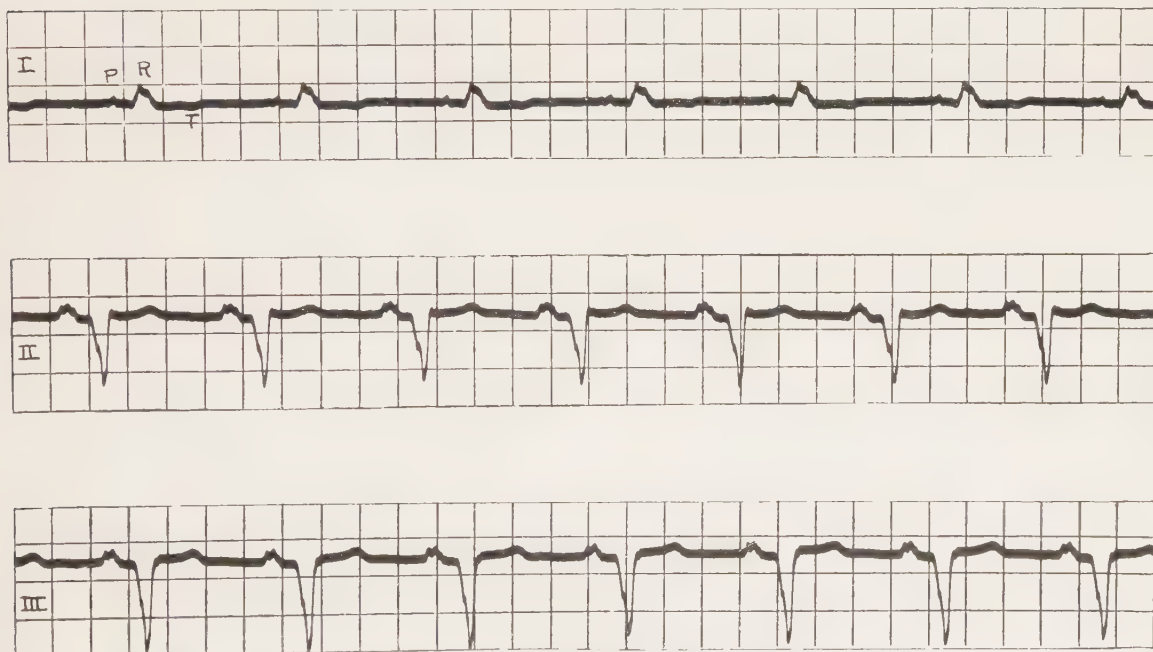


FIG. 154.—Record showing incomplete bundle-branch block. The QRS complexes are notched in all leads, but they exhibit greater deformity in lead I where their amplitude is unusually low. The T waves in lead I are inverted, and the P waves in leads II and III display distinct notching. Record obtained from a man, aged sixty-nine years, with coronary sclerosis.

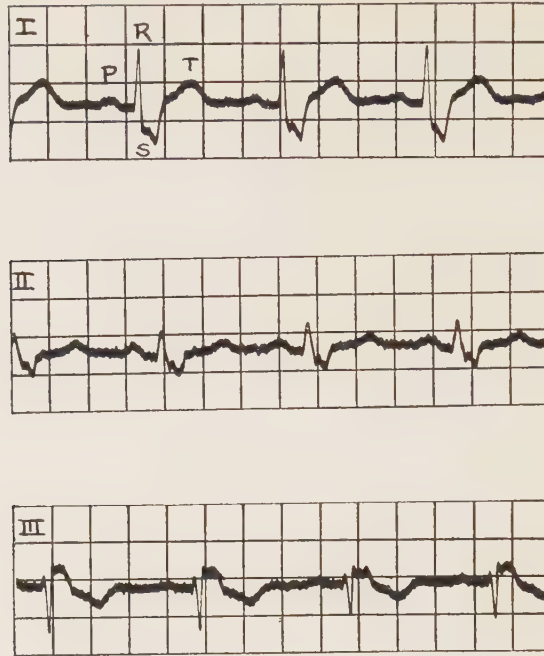


FIG. 155.—Record showing incomplete bundle-branch block. The notching of the QRS complexes involves the terminal portions in all leads. The T waves in lead III are inverted. Record obtained from a man, aged fifty-four years, with hypertensive heart disease.



FIG. 156.—Record showing incomplete bundle-branch block associated with delayed auriculoventricular conduction. The P-R interval is 0.28 second and the base width of the QRS complexes is 0.14 second. Deformity in contour of the QRS complexes is most marked in lead I. The T waves in lead I are diphasic. Record obtained from a man, aged seventy-three years, with hypertensive and coronary heart disease.

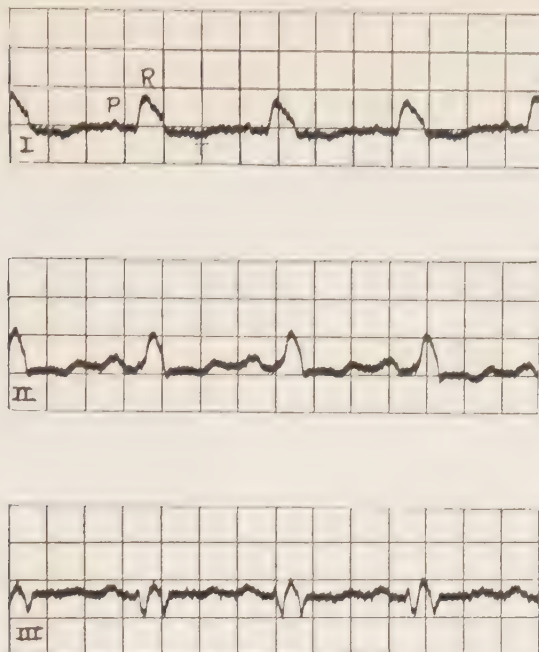


FIG. 157.—Record showing incomplete bundle-branch block with marked prolongation of the base width of the QRS complexes—0.18 second. The complexes are greatly abnormal in contour but are of relatively low amplitude. Note, in the QRS complexes in lead III, the peculiar notching which conforms to the so-called "W" complexes. The T waves in leads I and II are inverted. Record obtained from a woman, aged fifty years, with hyperfunctioning adenomatous goiter.

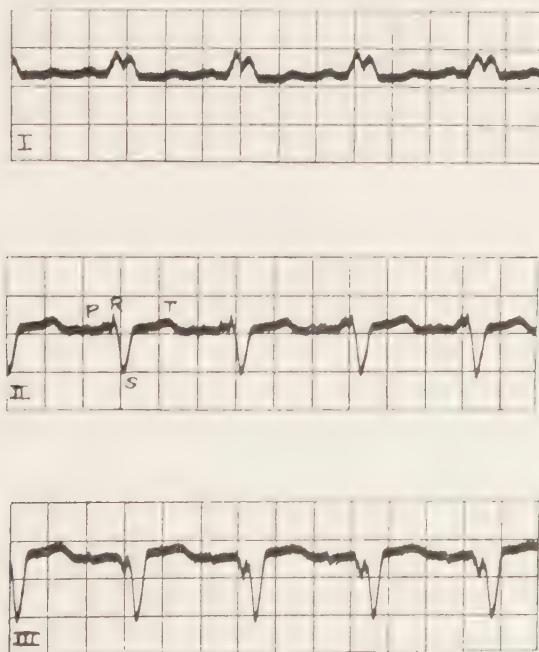


FIG. 158.—Record showing incomplete bundle-branch block. The QRS complexes are bizarre in all leads but are of normal amplitude. The base width of the complexes is 0.16 second. Note, in lead I, the peculiar notching of the QRS complexes; this notching has led to the adoption of the descriptive term "M" complexes. Record obtained from a man, aged sixty-eight years, with coronary sclerosis.

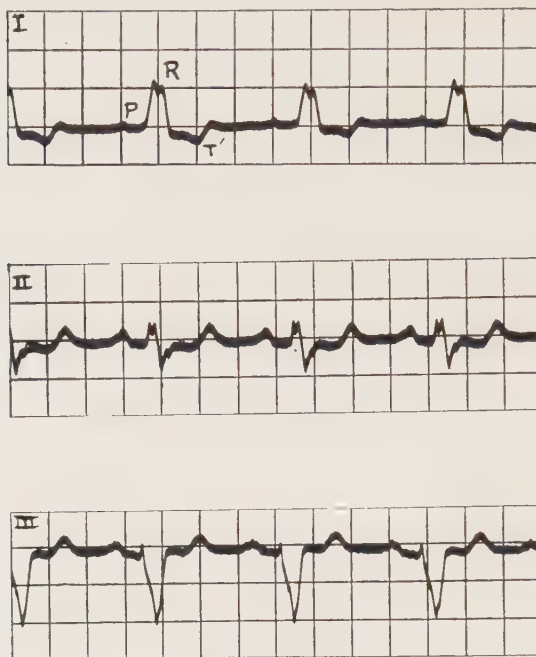


FIG. 159.—Record showing complete right bundle-branch block with complexes of somewhat lesser amplitude. The base width of the QRS complexes is 0.14 second, and notching occurs in all leads. The T component in lead I is directed downward. This record is somewhat transitional between those records of marked, incomplete bundle-branch block and those of complete bundle-branch block. Record obtained from a man, aged fifty-nine years, with coronary sclerosis accompanied by angina pectoris.

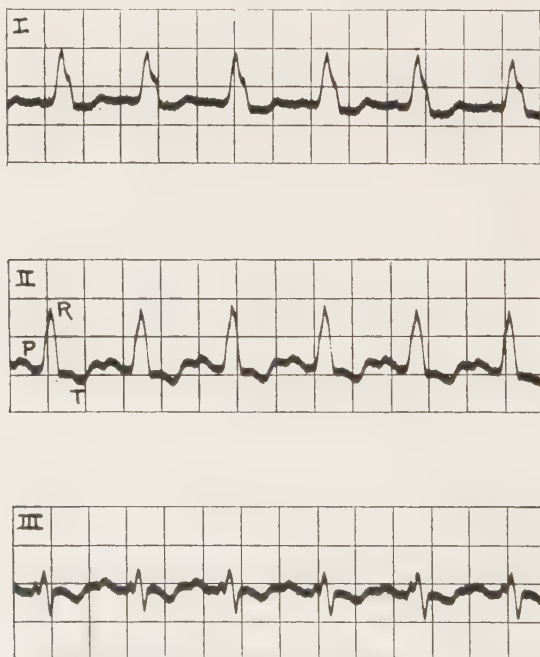


FIG. 160.—Record showing incomplete bundle-branch block. The base width of the QRS complexes varies from 0.11 to 0.12 second. Deformity of the complexes occurs in all leads. The T waves are diphasic in lead I and are inverted in leads II and III. There is slight preponderance of the left ventricle. Record obtained from a man, aged fifty-six years, with coronary sclerosis accompanied by angina pectoris.

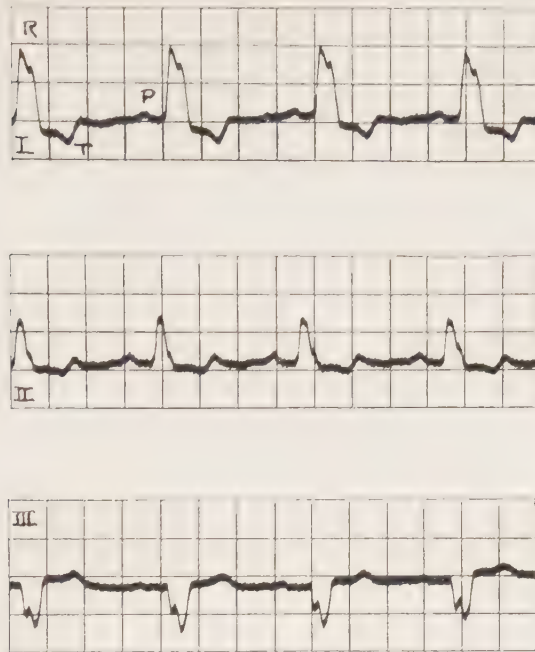


FIG. 161.—Record showing incomplete bundle-branch block. The QRS complexes show notching in all leads and the base width is 0.12 second. The T waves in lead I are inverted and in lead II are diphasic. Record obtained from a woman, aged forty years, with long-standing exophthalmic goiter.

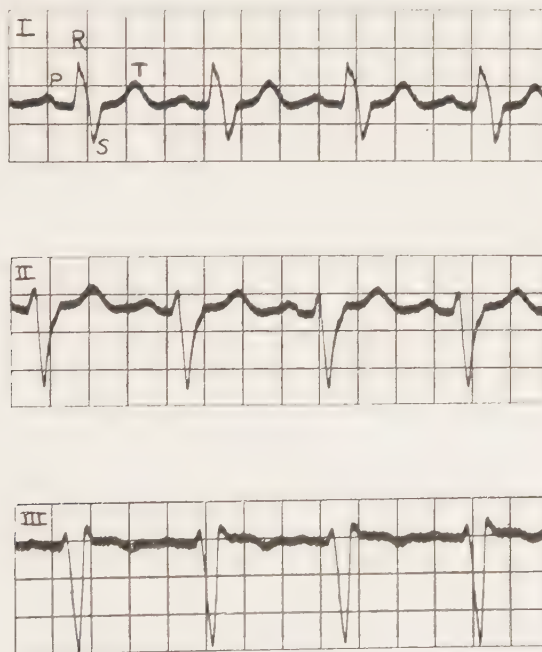


FIG. 162.—Record showing incomplete bundle-branch block. The base width of the QRS complexes ranges from 0.14 to 0.16 second. Slight deformity in the contour of the complexes occurs. The T waves in lead III are inverted. Record obtained from a man, aged fifty-five years, with coronary sclerosis.

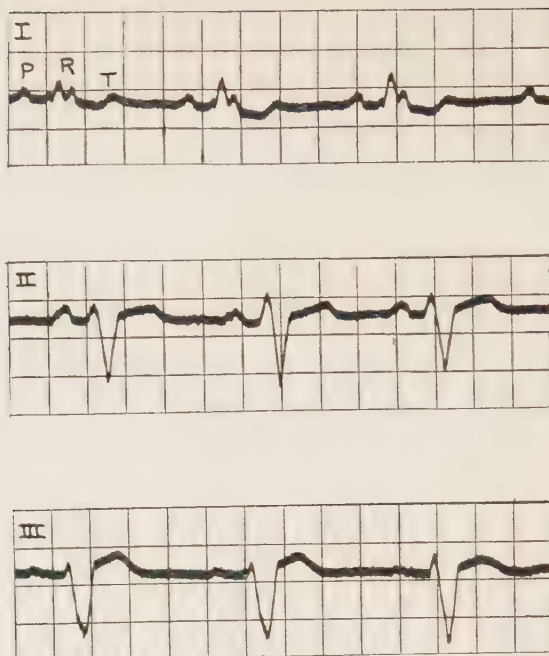


FIG. 163.—Record showing incomplete bundle-branch block. The base width of the QRS complexes is increased to 0.14 second. Marked deformity is limited to lead I where the complexes are of low amplitude. Record obtained from a man, aged fifty-seven years, with coronary sclerosis.

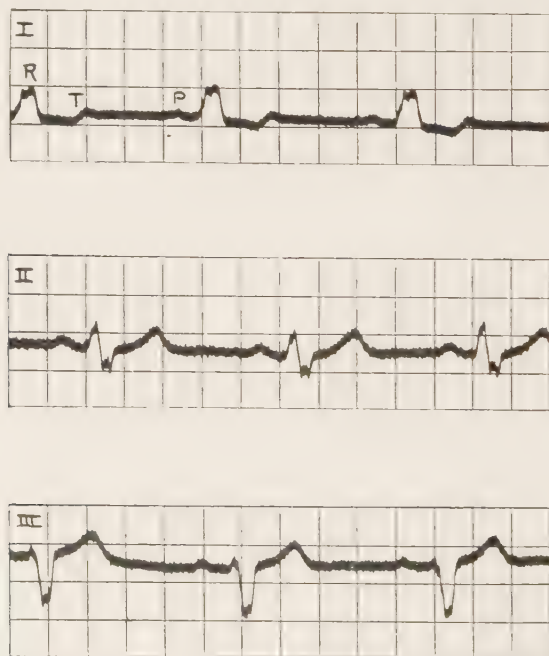


FIG. 164.—Record showing incomplete bundle-branch block. Rather marked notching of the QRS complexes occurs, in all leads, with increase in the base width—0.13 second. The T waves in lead I are inverted. Record obtained from a man, aged sixty-one years, with coronary sclerosis.

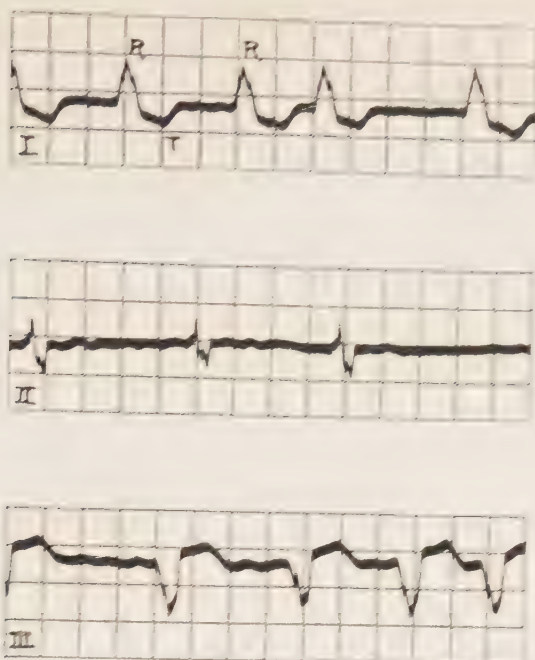


FIG. 165.—Record showing the coexistence of sinus bradycardia and incomplete bundle-branch block. The total irregularity is marked and the P waves are absent. The QRS complexes in all leads are abnormal in contour and their base width is increased (0.08 second). Record obtained from a woman, aged fifty-four years, with myocardial changes of long duration.

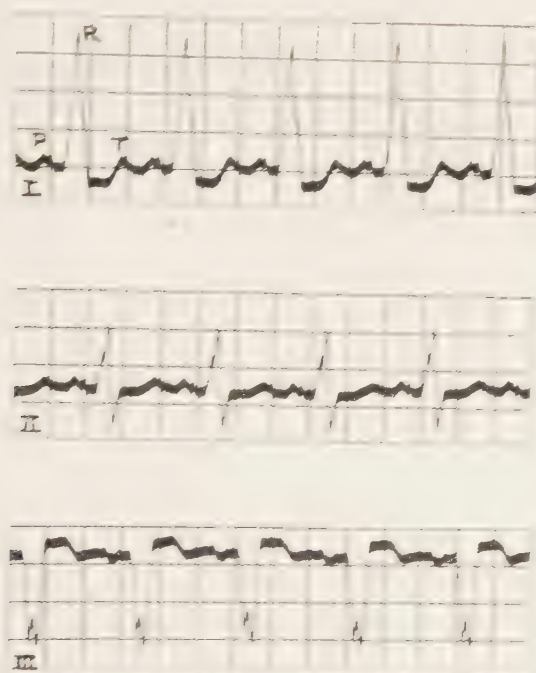


FIG. 166.—Record showing incomplete bundle-branch block. Considerable aberration of the QRS complexes is evident; the base width of the complexes is 0.12 second, and, especially in lead I, the amplitude is increased. Record obtained from a woman, aged sixty-five years, with coronary sclerosis.

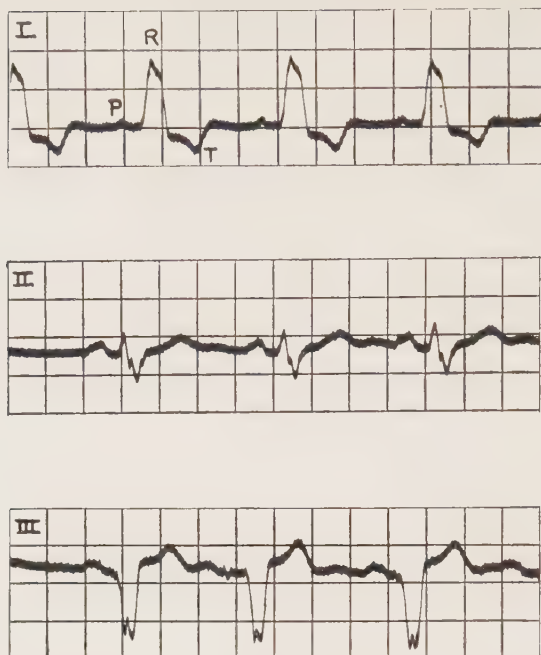


FIG. 167.—Record showing incomplete bundle-branch block of considerable degree. The base width of the QRS complexes is 0.14, 0.13, and 0.12 second. Distinct notching occurs in all leads. The T components in lead I are directed downward. Record obtained from a woman, aged sixty-seven years, with coronary sclerosis and hyperfunctioning adenomatous goiter.

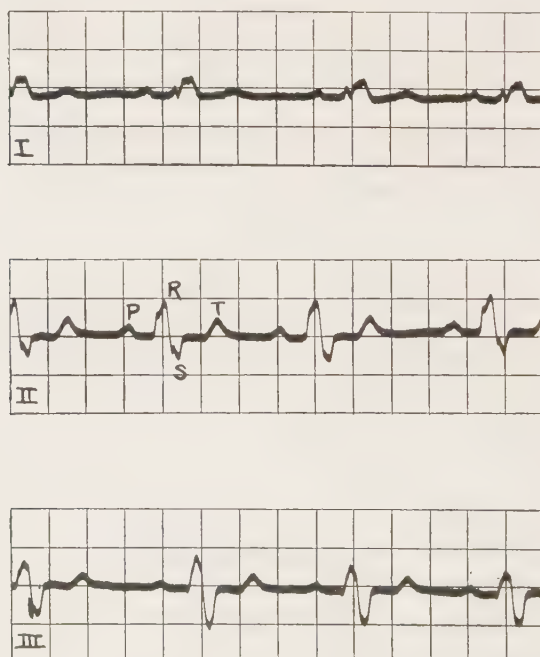


FIG. 168.—Record showing incomplete bundle-branch block. The QRS complexes are definitely deformed by notching in all leads but they are of rather low amplitude and have an increased base width—0.14 second. Record obtained from a man, aged twenty-nine years, with myocardial disease of indeterminate origin.

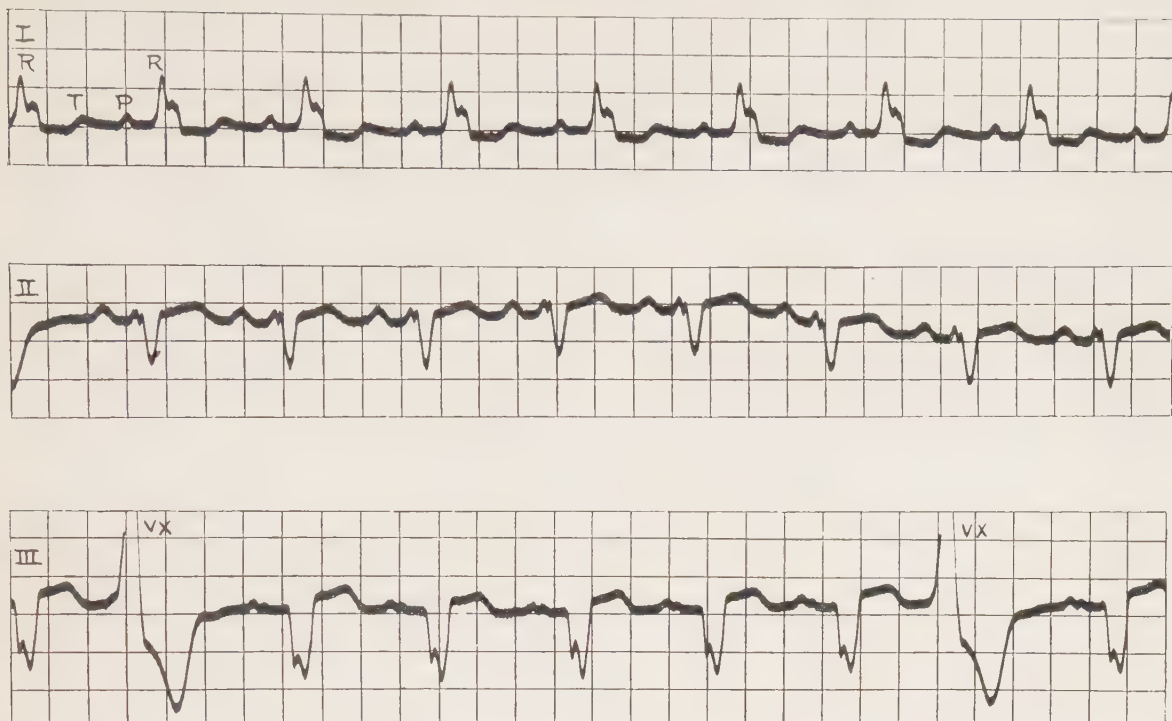


FIG. 169.—Record showing incomplete bundle-branch block of rather marked degree. The QRS complexes in all leads show notching which is especially marked in leads I and III. The base width is 0.15, 0.14, and 0.12 second in the respective leads. The T waves in lead I are diphasic; they have, however, an upright trend. Two premature ventricular contractions are present in lead III. Record obtained from a woman, aged sixty-six years, with coronary sclerosis accompanied by angina pectoris.

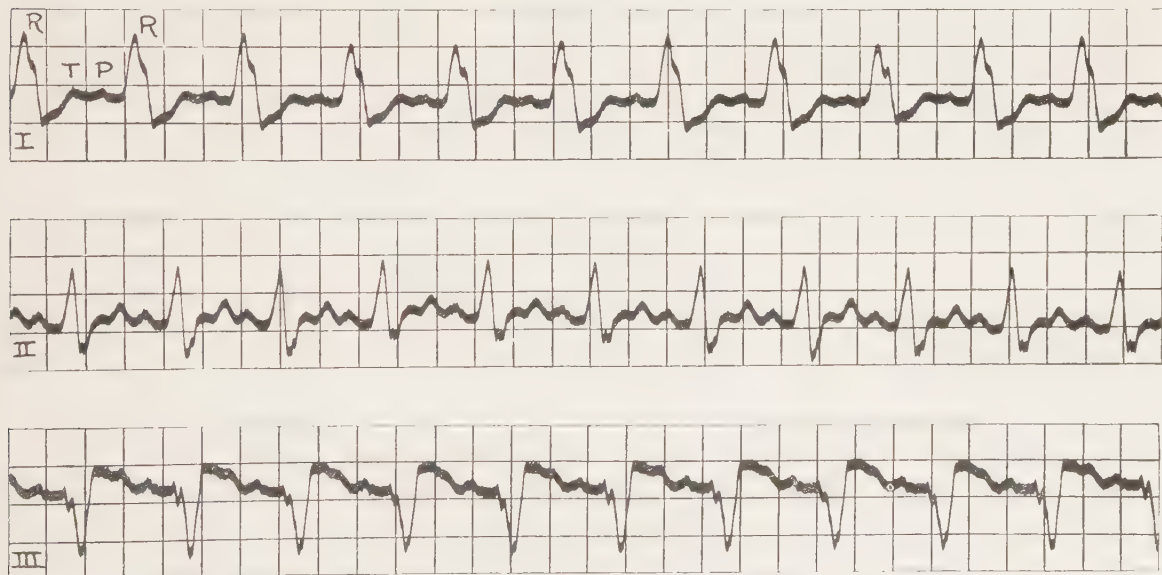


FIG. 170.—Record showing well marked, incomplete bundle-branch block. The QRS complexes are notched in all leads and the base width is considerably increased; namely, to 0.15 second. Distinct variations in the amplitude of the QRS complexes occur in lead II. Note the peculiar take-off of the T waves in leads I and III (coronary T wave). Record obtained from a man, aged sixty-six years, with coronary sclerosis accompanied by angina pectoris.

by the activation of the free wall of the right ventricle, which are oppositely directed, occur synchronously and tend to neutralize each other. In bundle-branch block these regions are not simultaneously activated, and therefore a disturbance of the normal balance between them takes place and relatively high complexes result.

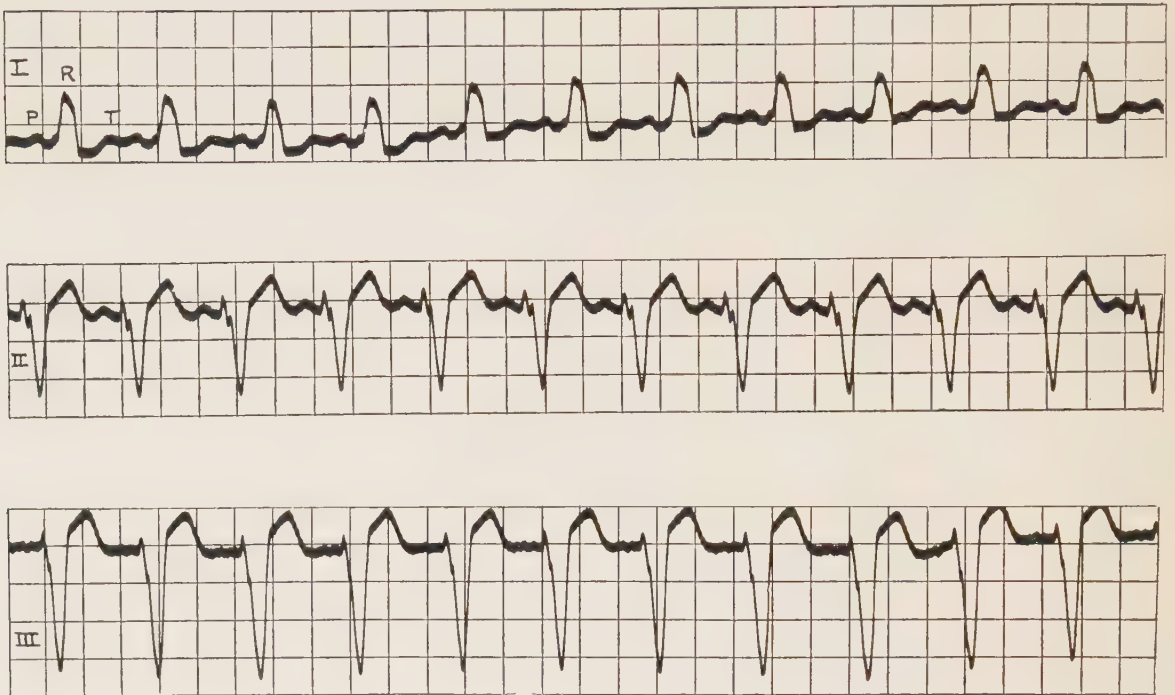


FIG. 171.—Record showing incomplete bundle-branch block of considerable degree; this record approaches in character the records of complete right bundle-branch block. The QRS complexes in lead III are of great amplitude, whereas those in leads I and II are considerably lower. The T waves in lead I are diphasic, with a trend toward the upright. The base width of the QRS complexes varies from 0.12 to 0.15 second in different leads. Distinct notching of the QRS complexes occurs in all leads. Record obtained from a woman, aged fifty-six years, with coronary sclerosis accompanied by angina pectoris.

In complete bundle-branch block, involving either branch, the T wave is exaggerated and its deflection is opposite to that of the main deflection of the QRS group. In clinical electrocardiograms of complete right bundle-branch block, the QRS complexes are directed

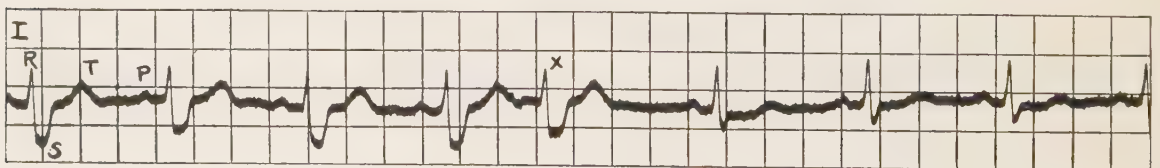


FIG. 172.—Record in lead I showing transient incomplete bundle-branch block. Note the deformity of the QRS complexes which subsequently return to normal. A premature contraction precedes the establishment of normal rhythm. Record obtained from a woman, aged sixty-eight years, with essential hypertension.

upward in lead I, whereas the T waves are directed downward, and the reverse occurs in lead III. The complexes in lead II may resemble either lead I or lead III. In clinical electrocardiograms of complete left bundle-branch block the QRS complexes are directed

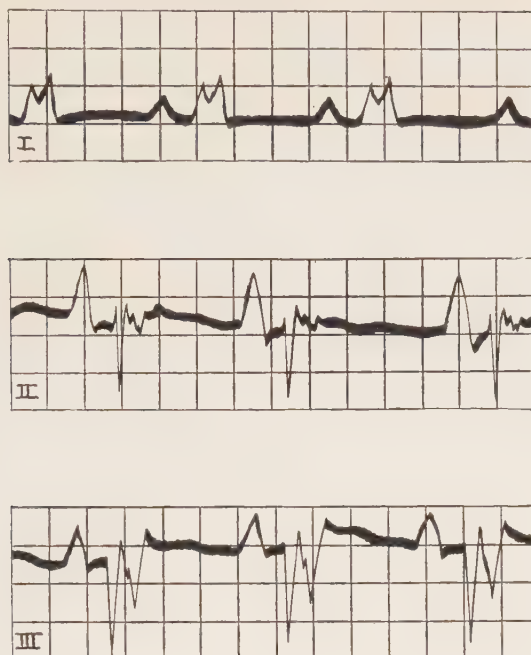


FIG. 173.—The record in this figure and in Figure 174 were obtained from the same patient on two successive days. Incomplete bundle-branch block is present. This gives rise to extremely bizarre notching of the QRS complexes and to P waves of unusually high amplitude and peculiar contour in leads I and II. The T waves in lead I are iso-electric. Record obtained from a woman, aged twenty-four years, who had chronic endocarditis with mitral stenosis associated with paroxysms of tachycardia and accompanied by pulmonary edema.**

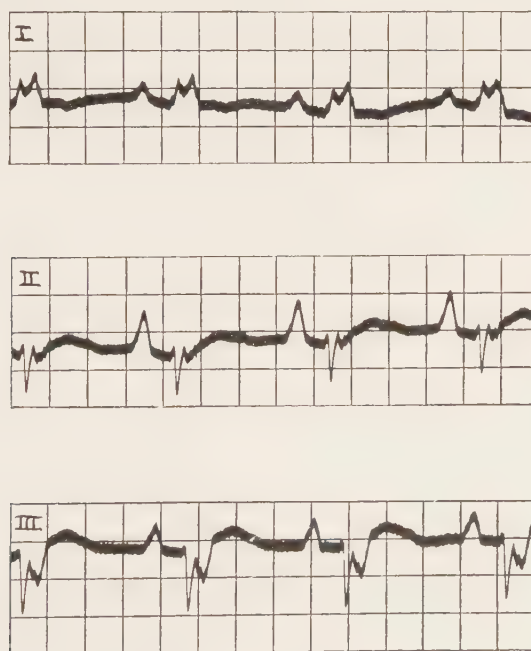


FIG. 174.—Record obtained from the same patient (Fig. 173) on the following day. The differences in the two records are apparent. Note the inversion of the T waves in lead I.

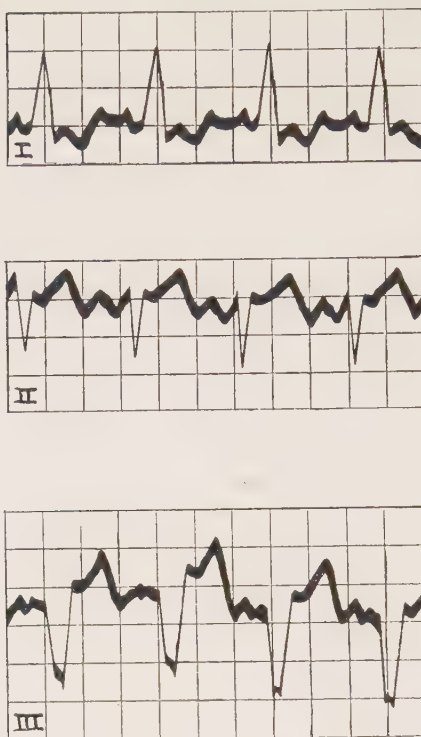


FIG. 175.—Record showing intermittent, incomplete bundle-branch block. This record was taken very soon after the onset of acute pulmonary edema and during severe heart failure. Record obtained from a woman, aged sixty years, with coronary sclerosis.**

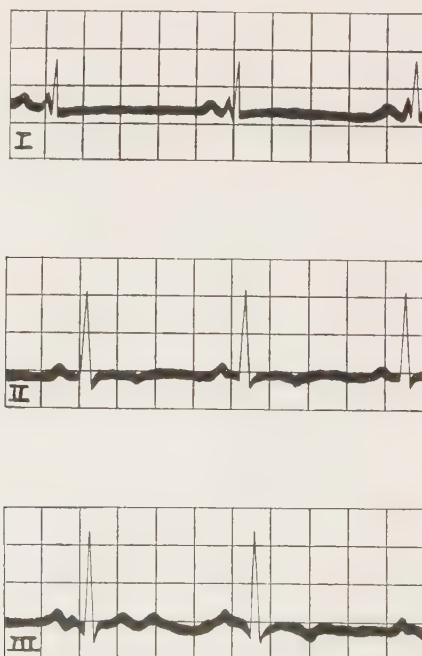


FIG. 176.—Record of the same patient (Fig. 175) taken seven days later than the previous record and showing the disappearance of incomplete bundle-branch block. The T waves in leads II and III are inverted and the T waves in lead I are iso-electric.**

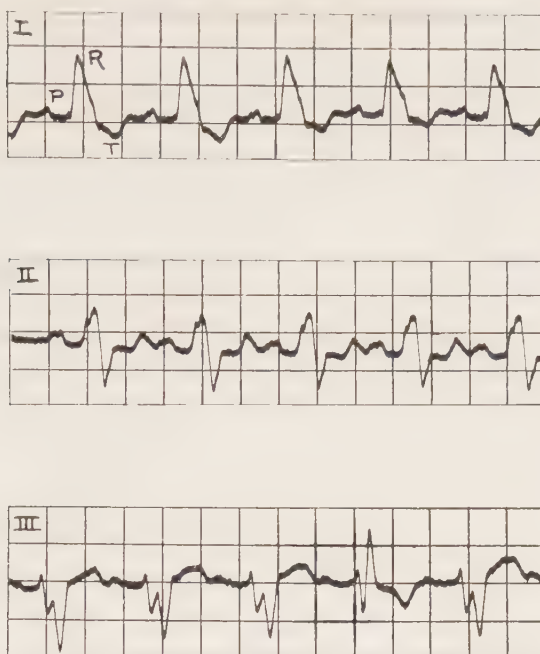


FIG. 177. —Record showing complete right bundle-branch block with marked deformity of the QRS complexes. The base width of the complexes is 0.14 second. The T components in lead I are directed downward. Note the interpolated premature ventricular contraction in lead III. Record obtained from a man, aged sixty-eight years, with hypertensive heart disease.

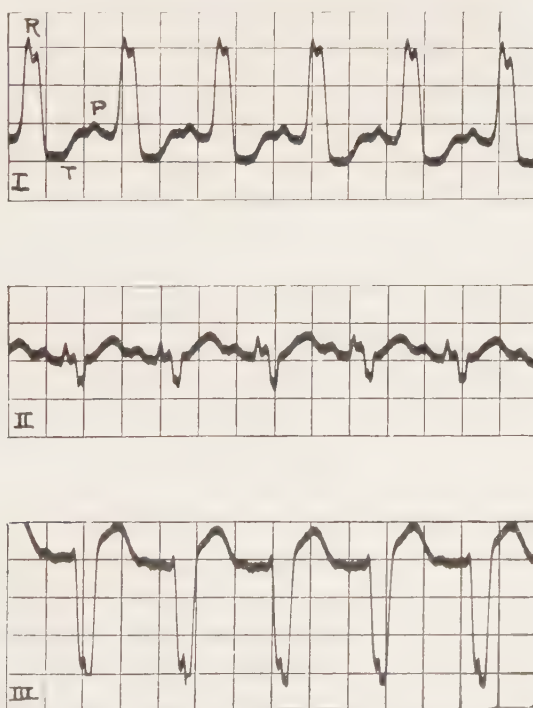


FIG. 178. —Record showing complete right bundle-branch block. The QRS complexes are notched in all leads and exhibit high amplitudes in leads I and III. The base width of the complexes is 0.14 second. The T waves in lead I are of the steep, diphasic type. Record obtained from a woman, aged sixty-seven years, with hypertensive and coronary heart disease and hyperfunctioning adenomatous goiter.

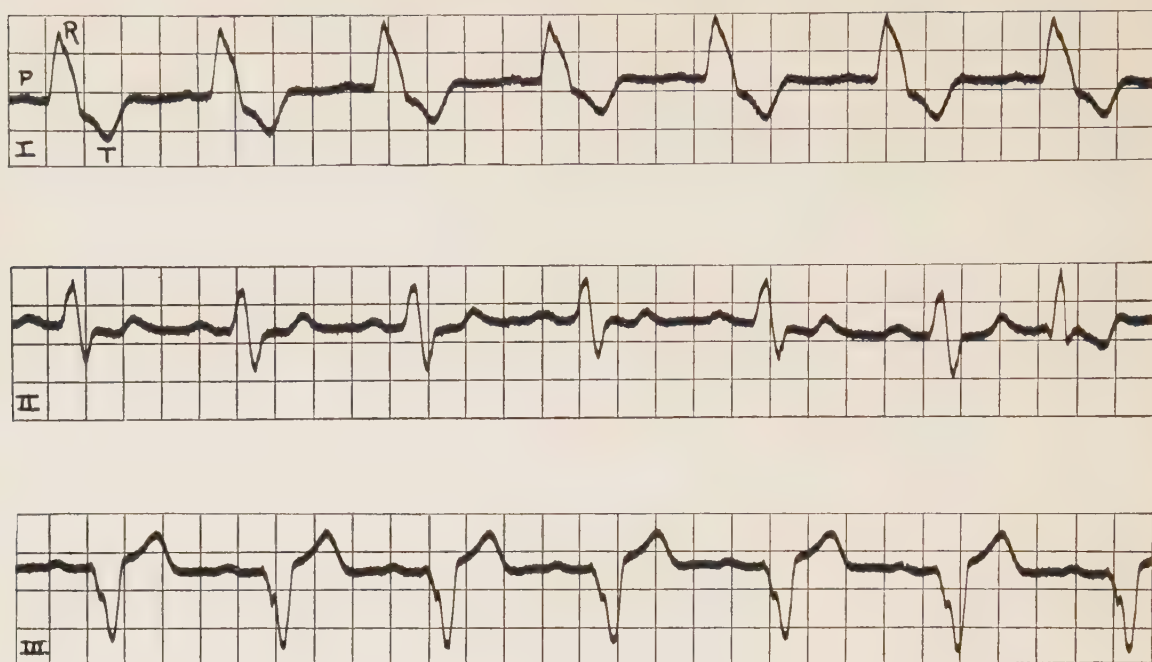


FIG. 179.—Record showing complete right bundle-branch block. Marked deformity of the QRS complexes occurs in all leads. The complexes are of high amplitude, with base width of 0.16 second. The T component in lead I is directed downward. Record obtained from a man, aged sixty years, with coronary sclerosis accompanied by angina pectoris.

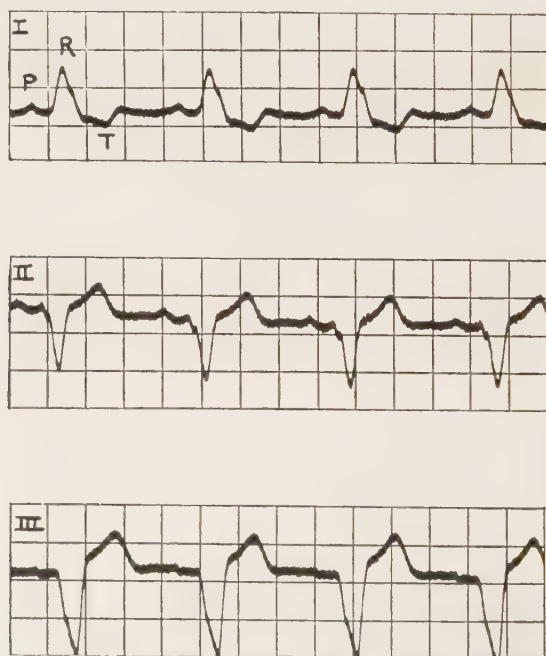


FIG. 180.—Record showing complete right bundle-branch block. The QRS complexes are abnormally wide, 0.14 to 0.16 second, with notching in all leads. The T components in lead I are directed downward. Record obtained from a man, aged fifty years, with hypertensive and coronary heart disease.

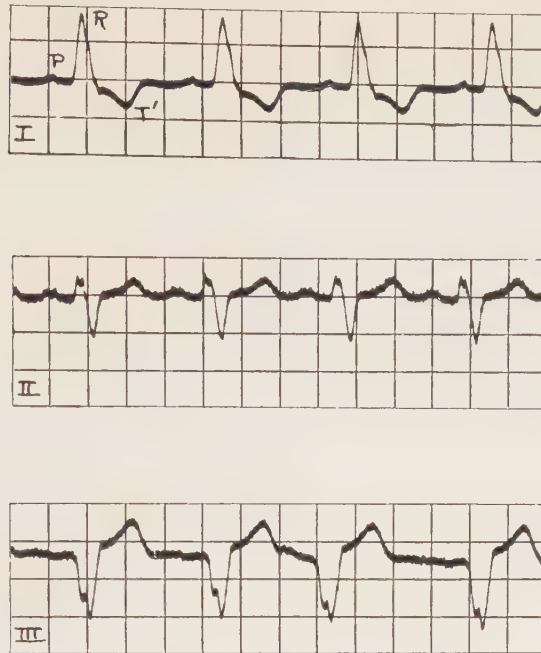


FIG. 181.—Record showing complete right bundle-branch block. The QRS complexes are of increased amplitude, of abnormal contour, and, in all leads, exhibit a base width of 0.14 second. The T component is directed downward in lead I. Record obtained from a man, aged fifty-five years, with advanced hypertensive and coronary heart disease.

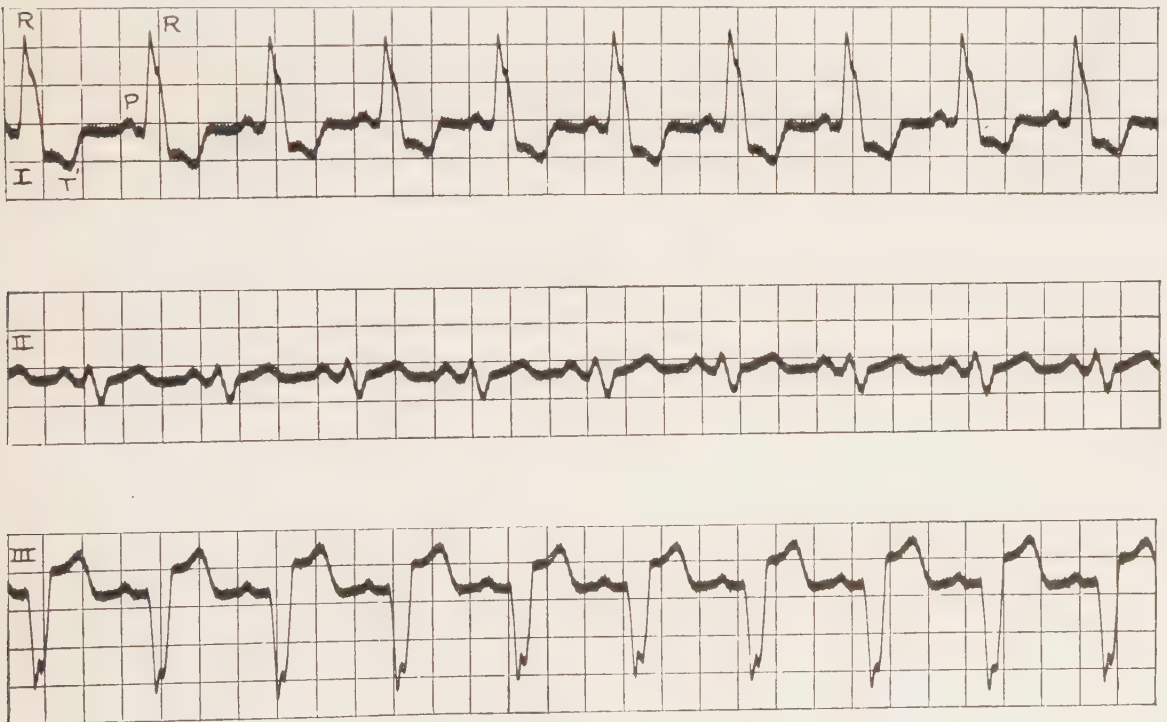


FIG. 182.—Record showing complete right bundle-branch block. The QRS complexes are bizarre in all leads, with increased amplitude in leads I and III, and the base width changes from 0.12 to 0.13 second. The T components in lead I are directed downward. Record obtained from a woman, aged fifty-four years, with coronary and hypertensive heart disease.

downward in lead I, whereas the T waves are directed upward; the reverse occurs in lead III. Notching of the components of the QRS complexes usually is present.

Before referring to the figures illustrative of complete right and left bundle-branch block, I wish to discuss those electrocardiograms that are similar to but not identical with those already described. In many clinical records the amplitude of deflection of the QRS complexes is less and the QRS intervals are considerably less than those which conform to the characteristics of complete bundle-branch block. In some earlier publications such graphs were attributed to defective intraventricular conduction, and in others, to disease of the Purkinje plexus.

Smith, in his early experimental work, ligated various branches of the coronary artery in the dog. He found that extensive lesions of the subendocardial myocardium occurred

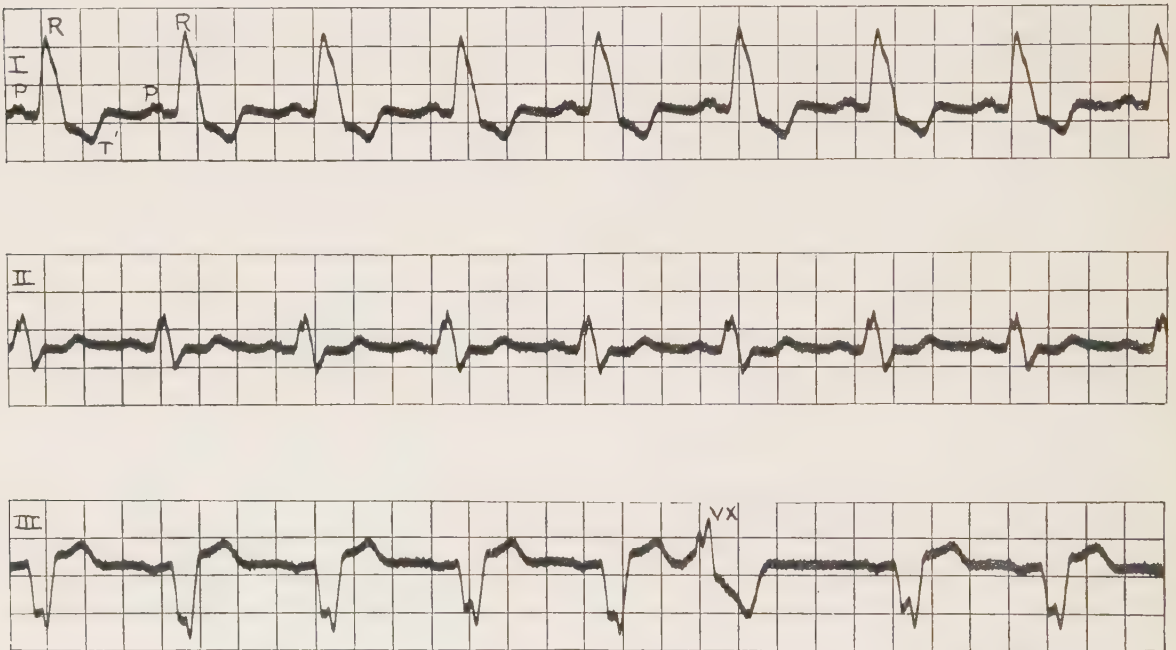


FIG. 183.—Record showing complete right bundle-branch block. The QRS complexes are bizarre in all leads and are of fairly high amplitude in leads I and III. The base width of the complexes is 0.12 second. The T component is directed downward in lead I. A premature ventricular contraction occurs in lead III. Record obtained from a woman, aged sixty-eight years, with coronary sclerosis.

but that changes in the QRS complexes of the electrocardiogram did not result. In later experiments he was able to produce QRS changes by subjecting the heart to unusual strain, and, at the same time, incising the endocardium. This led him to conclude that at least two factors were necessary for the production of abnormal QRS complexes; namely, lesions of the conduction system and cardiac fatigue.

The complexes under discussion were grouped by Wilson and Herrmann as transitional between normal complexes and those of complete bundle-branch block and were called incomplete bundle-branch block. Their studies led them to conclude that these transitional electrocardiograms resulted from delay in transmission of the impulses through one of the main branches of the auriculoventricular bundle. Owing to changes in position

of the heart, to individual characteristics of the architecture of the ventricular muscle, and to modifications of the Purkinje system, variations occur in the QRS complexes in normal hearts. Accordingly, caution must be exercised in the interpretation of minor aberrations. Under conditions of disease, still further variations in the abnormal complexes of complete and incomplete bundle-branch block may occur.

Numerous examples of incomplete bundle-branch block, showing many variations, are presented (Figs. 150 to 176). Not infrequently it is difficult to decide whether an electrocardiogram represents an instance of rather marked incomplete bundle-branch block or of complete bundle-branch block.

In Figure 150 definite but rather slight changes in the QRS complexes in all leads occur. The deformity of the QRS complexes is not extreme but the duration of the com-

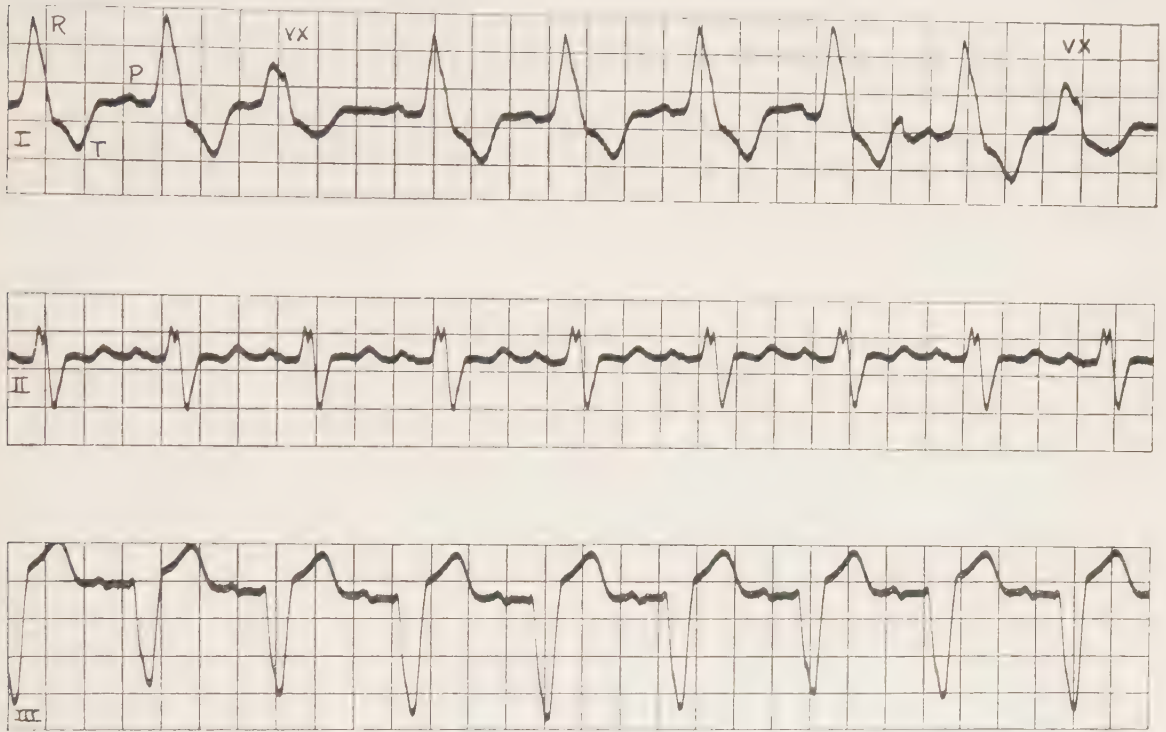


FIG. 184. Record showing complete right bundle-branch block. The complexes, which are of high amplitude, are deformed by slurring and notching; they display a greatly increased base width, varying, in different leads, from 0.16 to 0.18 second. The T components are directed downward in lead I and upward in lead III. In lead I two premature ventricular contractions occur which are identified by their prematurity and change in contour. Record obtained from a man, aged fifty-seven years, with syphilitic aortitis.

plexes is increased to 0.12 second. Such a record is classed as incomplete bundle-branch block. The changes in Figures 151 to 160 are more marked. There is some question as to whether such graphs as those shown in Figures 159 and 167 are evidence of incomplete or complete bundle-branch block, and the interpretation may vary owing to the personal equation.

Incomplete bundle-branch block may be associated with other graphic abnormalities. In Figure 156 is shown its association with delayed auriculoventricular conduction; in Figure 165, with auricular fibrillation, and in Figures 77 and 79, with auricular flutter.

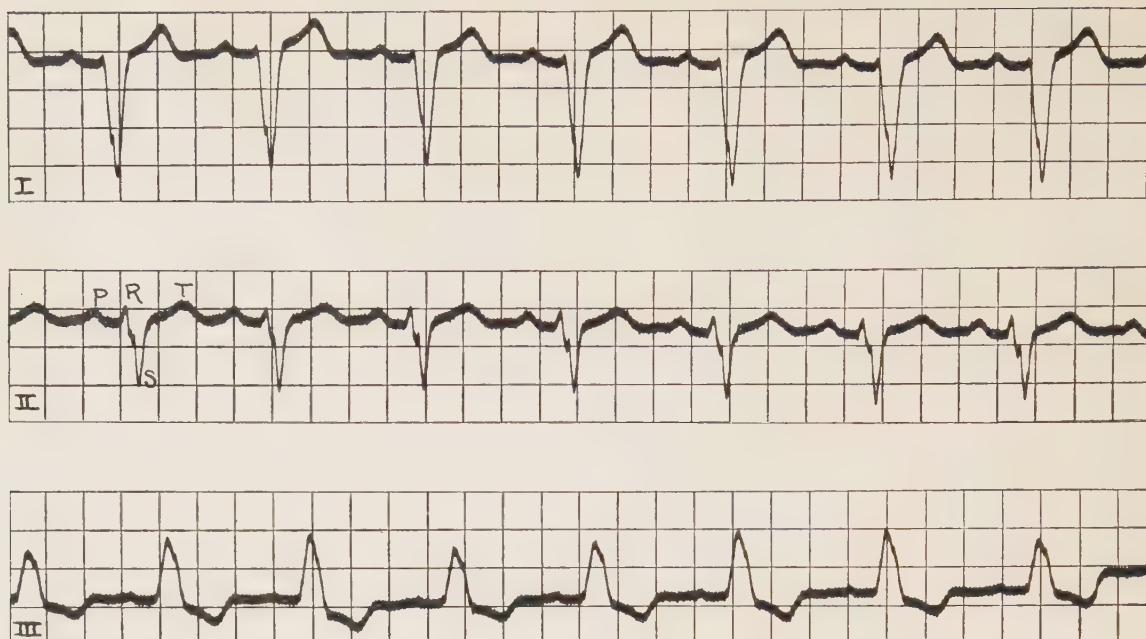


FIG. 185.—Record showing complete left bundle-branch block. Note that this record is the mirror image of those displaying complete right bundle-branch block. In lead I the QRS complexes are directed downward and the T components are upright; in lead III, the QRS complexes are directed upward and the T components downward. The amplitude of the complexes is increased, especially in lead I, and the base width of the QRS complexes is 0.15 second. Record obtained from a man, aged fifty-nine years, with coronary and hypertensive heart disease.

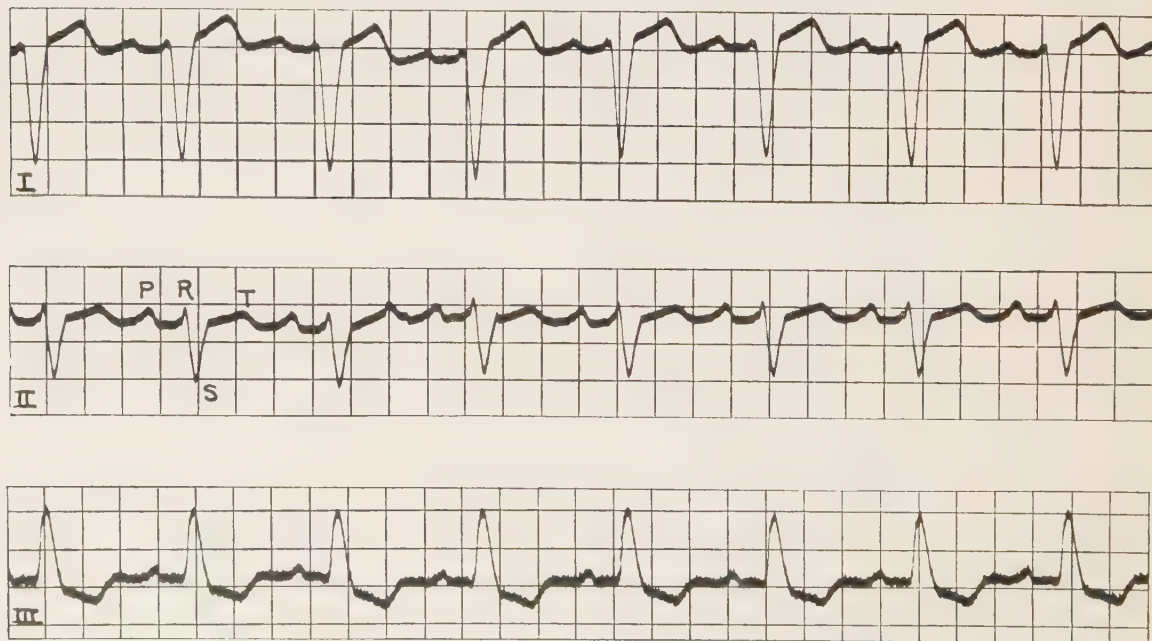


FIG. 186.—Record showing complete left bundle-branch block. Note the bizarre QRS complexes: the base width varies from 0.14 to 0.16 second and the deep complexes in lead I are directed downward. The QRS complexes in lead III are directed upward, whereas the T components are directed downward. Record obtained from a man, aged sixty-two years, with coronary and hypertensive heart disease.

Incomplete bundle-branch block is occasionally present as a transient disorder, as seen in Figures 172 to 176.

In the interpretation of these electrocardiograms, caution must be used in definitely linking them with actual lesions of the conduction system. It is probable that marked interference in the function of the conduction system may exist without the presence of actual histopathologic changes. This is probably true in coronary disease, in which nutritional effects are in evidence; it may occur, during the stage of failure, from many causes; it may be a phenomenon of fatigue, or it may occur in greatly hypertrophied hearts in which impairment of conduction, without actual disease of the conduction paths, occurs.

Incomplete bundle-branch block is most frequently observed in coronary and hypertensive heart disease. It has been observed, with less frequency, in other cardiopathies.

It is a significant graphic abnormality and furnishes reliable evidence toward an unfavorable prognosis. Table 11 shows the statistical data in 237 cases of incomplete bundle-branch block. The figures are appalling in their revelation that 154 patients

TABLE 11
MORTALITY IN CASES OF INCOMPLETE BUNDLE-BRANCH BLOCK

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
10-19	12	7	5	3	25	15		9	75
20-29	21	15	6	15	71	21	2	4	19
30-39	19	10	9	14	74	12	1	4	21
40-49	57	38	19	30	53	16	3	24	42
50-59	68	44	24	42	62	14	2	24	35
60-69	45	33	12	39	87	9	2	4	9
70-79	15	10	5	11	73	6	1	3	20
Total and average.	237	157	80	154	65	13	11	72	30

(65 per cent) died of heart disease in an average of thirteen months following examination. Almost identical percentages have been obtained in previous follow-up studies.

The electrocardiograms of complete bundle-branch block are more distinct owing to the greater amplitude of deflection and the greater period of duration of the QRS complexes. These records so closely resemble those produced in experiments in which one or the other main bundle-branch was actually divided that they are highly suggestive of an actual lesion. However, it is not impossible that the hypothetic influences discussed under incomplete bundle-branch block may be active here.

Complete right bundle-branch block is much more common than complete left bundle-branch block. In the material at The Mayo Clinic the incidence of the two types was: complete right bundle-branch block, 94 per cent; complete left bundle-branch block, 6 per cent. This difference in incidence has been ascribed to anatomic differences between the two branches. The right branch passes, undivided, directly to the large papillary muscle

of the right ventricle; the left branch divides almost immediately and spreads out in a fan-like manner, thus rendering complete obstruction unlikely except when a very extensive lesion is present. Furthermore, judging from unconfirmed anatomic studies of the terminal coronary arteries, the left bundle appears to have a dual blood supply, whereas the right appears to have only one supplying branch. If these observations are confirmed, a probable explanation of the discrepancy in incidence is at hand.

Typical examples of complete right bundle-branch block are shown in Figures 177 to 184 and of complete left bundle-branch block in Figures 185 and 186.

I have never observed complete bundle-branch block as a temporary or transient disorder. It occurs in the same types of cardiopathy in which incomplete bundle-branch block occurs and in about the same relative proportion. Likewise it has about the same prognostic importance as incomplete bundle-branch block.

Table 12 contains the mortality statistics of eighty-six cases of complete bundle-branch block. Fifty-seven patients (66 per cent) died of heart disease within an average of twelve months following examination. There is apparently little difference in significance between the electrocardiograms of right and of left complete bundle-branch block.

TABLE 12
MORTALITY IN CASES OF COMPLETE BUNDLE-BRANCH BLOCK

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
20-29	3	2	1	1	33	19		2	67
30-39	2	1	1	1	50	14		1	50
40-49	7	5	2	5	71	10	1	1	14
50-59	33	19	14	22	67	17	2	9	28
60-69	34	23	11	23	68	9	2	9	26
70-79	7	5	2	5	71	4	1	1	14
Total and average.	86	55	31	57	66	12	6	23	17

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Chapter X

SINO-AURICULAR BLOCK

Sino-auricular block is a rare disturbance of cardiac action. It consists of dropped beats and represents periods of complete cardiac standstill. The auricle may fail to contract, and the ventricle subsequently may fail to beat because (1) an impulse did not form, because (2) the auricle was temporarily unable to respond to an impulse that might have been either adequate or inadequate, or because (3) the impulse was blocked between the sino-auricular node and the auricle. During the period of sino-auricular block, the rhythmic

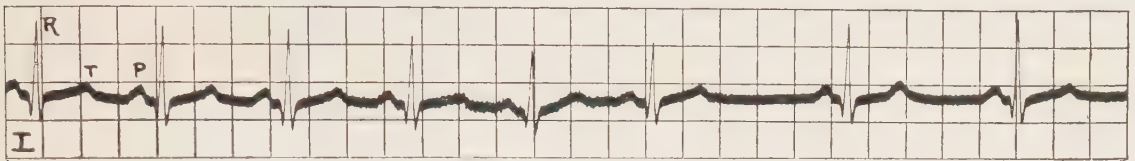


FIG. 187.—Record in lead I showing sino-auricular block. There is complete cardiac standstill for one cycle; one beat of the last two complexes is dropped out. The interval of these long cycles is a little less than the time occupied by two normal cycles. Record obtained from a woman, aged fifty-one years, with essential hypertension.

action of both auricles and ventricles is interrupted by a cycle of unusual length. The cycle is usually slightly less than the interval comprising two normal beats. Occasionally, two or three beats may be omitted during the period of block. Except for the interruption in rhythm, the electrocardiogram remains unaltered: the individual waves are normal in

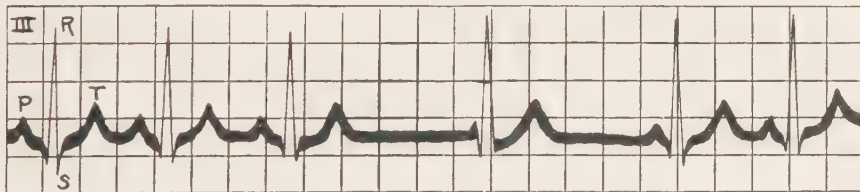


FIG. 188.—Record in lead III showing sino-auricular block. Note that the long cycles are a trifle less than the length of two normal cycles. Record obtained from a boy, aged twelve years, without evidence of organic heart disease.**

contour and maintain their proper relations in time to one another (Figs. 74, 75, 187, and 188).

This disorder is presumed to result from abnormal, inhibitory vagal influences and is frequently a transitory phenomenon. The administration of atropine often abolishes the disorder. It has been observed during the administration of digitalis, aconitine, salicylic acid, morphine, and in the excessive use of tobacco. The condition is usually observed in adults, but has been recognized in young children. As a rule, sino-auricular block has little clinical significance.

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Chapter XI

PAROXYSMAL TACHYCARDIA

Paroxysmal tachycardia, in the strict sense of the term, comprises three forms of cardiac acceleration which arise from the production of abnormal stimuli in the ventricles, in the auricles, or in the auriculoventricular junctional tissues. The origin of the impulse in these forms of tachycardia is ectopic; that is, in some area of the heart away from the sino-auricular node. Lewis insists that the term "paroxysmal tachycardia" be reserved for those forms of tachycardia which are of ectopic origin since the graphic complexes which represent paroxysmal tachycardia and those which represent a series of premature contractions are identical in appearance.

Therefore, perhaps other forms of paroxysmal cardiac acceleration are best considered independently.

Paroxysmal tachycardia is characterized by the sudden inception of an extremely rapid rhythm, of variable duration, and usually of abrupt termination.

Experimentally, paroxysmal tachycardia has been produced in numerous ways. Faradization has been employed. The phenomenon has been observed following the application of a clamp to the aorta, with resulting rise in ventricular pressure. Following ligation of branches of the coronary artery, with resulting myocardial ischemia, paroxysms of tachycardia have been noted. The administration of drugs, intravenously and otherwise, has resulted in these disturbances of cardiac action; notably, digitalis, strophanthine, chloroform, nicotine, epinephrine, ether, pituitary extract, potassium and barium salts, and aconitine have caused tachycardia.

The condition as it occurs in man is still not fully understood; and, owing to the lack of definite data, the causative disturbance is perhaps best called, rather inadequately, "increased cardiac irritability."

The influence on disturbed cardiac action of the extrinsic cardiac nerves (vagus and sympathetic nerves) has been investigated by many workers. In paroxysmal tachycardia the observation repeatedly has been made that at times a paroxysm may be suddenly arrested by vagal pressure. Statements regarding other influences of innervation are hypothetical. Lewis believes that the abolition of vagal tone may be a factor in the production of a paroxysm of tachycardia but that other factors are also at play in rendering the heart more susceptible to stimuli.

The belief has been advanced that excitation of the accelerator nerves probably takes place in the intracardiac ganglia, and that this excitation is an important influence in the production of paroxysmal tachycardia.

Certain clinical observations are of interest in this connection, although they lack scientific confirmation. The fact that the attacks are frequently precipitated by emotional disturbances, their occurrence at times in individuals of unstable nervous make-up, and their frequency in cases in which repeated examination fails to disclose evidence of cardiac disease all suggest the presence of a neurogenic factor in the etiology. Histopathologic studies of material from patients with these disorders have been few and unsatisfactory.

In the report of a case of paroxysmal tachycardia recently published by Graber the vagi were found to be degenerated as the result of pressure exerted by enlarged mediastinal nodes. The primary condition in the illness of this patient had been Hodgkin's disease.

Paroxysmal tachycardia is most frequently observed in adults, although authentic instances have been reported in infants and children. The prognosis seems to depend chiefly on the type and extent of the associated cardiac disease rather than on the frequency, duration, and other features of the tachycardia.

VENTRICULAR TACHYCARDIA

Ventricular tachycardia is a rather rare disorder of cardiac mechanism and its infrequent occurrence affords meager information regarding its etiology. In a few cases in which the heart was subjected to careful pathologic study, obliterative sclerosis of the left coronary artery was demonstrated. This observation, together with the experimental pro-

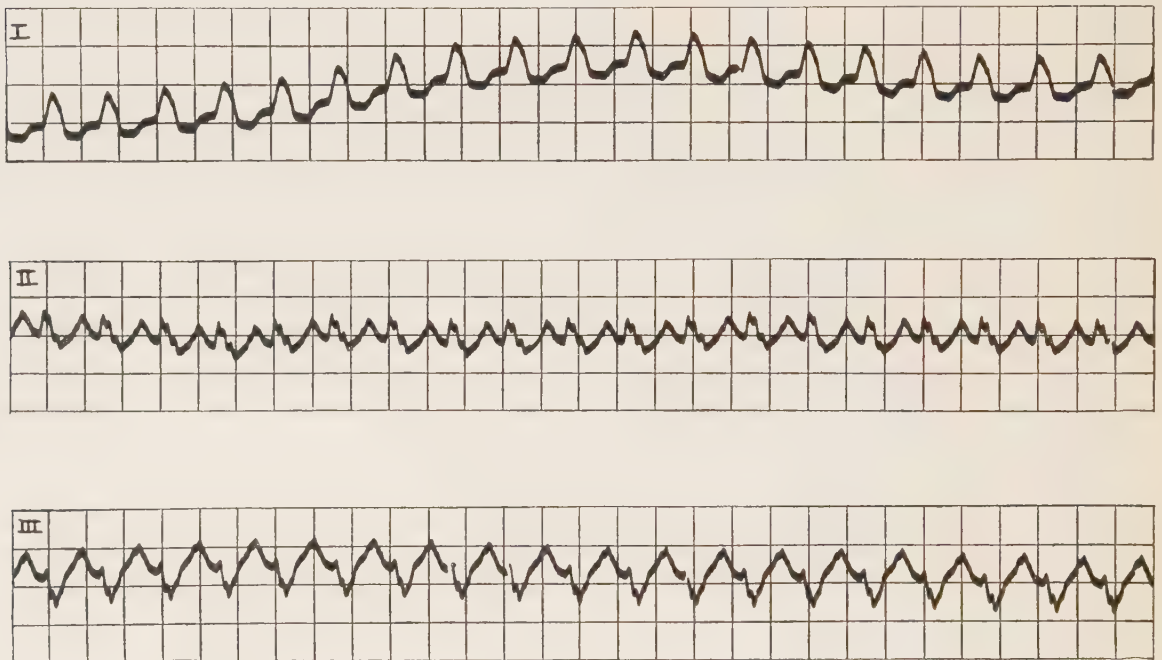


FIG. 189.—Record showing a paroxysm of ventricular tachycardia; the rate is 195 contractions each minute. The record is a rapid succession of premature ventricular contractions. The amplitude of the complexes is unusually low. Record obtained from a man, aged fifty-nine years, with coronary sclerosis accompanied by angina pectoris.

duction of ventricular tachycardia by means of ligation of the coronary artery, is suggestive of at least one etiologic factor. That other imposed conditions may also result in this condition is highly probable; clinically it has been observed as a toxic effect of digitalis, strophanthine and quinidine and experimentally it has been observed following the administration of chloroform and epinephrine. The area in which the ectopic or abnormal impulse is formed is in some portion of the ventricles.

The electrocardiograms exhibit successions of abnormal ventricular complexes, indistinguishable from those characteristic of ventricular premature contractions. The general contour of the complexes may vary considerably from case to case (Figs. 189 to 194).

Auricles and ventricles usually contract at the same rate, although an instance has been observed in which the auricular rate exceeded the ventricular rate. The ventricular rhythm

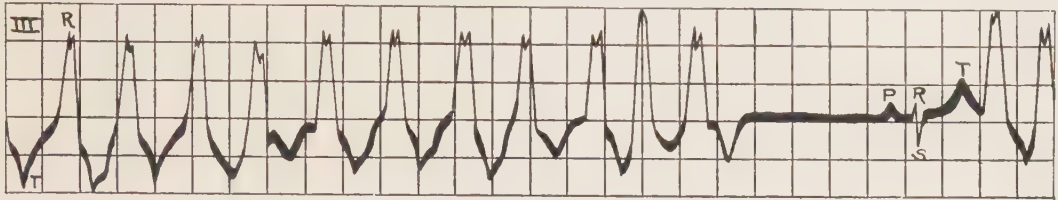


FIG. 190.—Record in lead III showing a paroxysm of ventricular tachycardia. Note the differences in character between the individual QRS complexes, which are but a succession of premature ventricular contractions. Note the transient restoration of normal rhythm. Record obtained from a man, aged fifty-six years, with coronary sclerosis accompanied by paroxysms of tachycardia. Necropsy revealed extensive coronary sclerosis with especial involvement of the left anterior descending branch of the artery.**

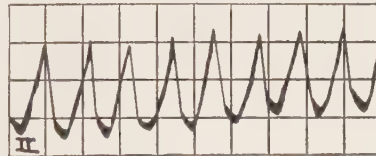


FIG. 191.—Record in lead II showing ventricular tachycardia. Note the series of bizarre QRS complexes. Record obtained from a man, aged thirty-four years, with slight cardiac hypertrophy of undetermined origin.**

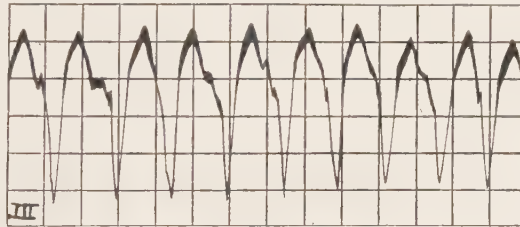


FIG. 192.—Record in lead III showing ventricular tachycardia. Note the high amplitude and the aberrant QRS complexes which in reality are serial premature ventricular contractions. Note the differences in contour between the individual complexes. Record obtained from a man, aged sixty-four years, with coronary sclerosis. Necropsy revealed almost complete atherosclerotic obliteration of the left anterior descending branch of the coronary artery.**

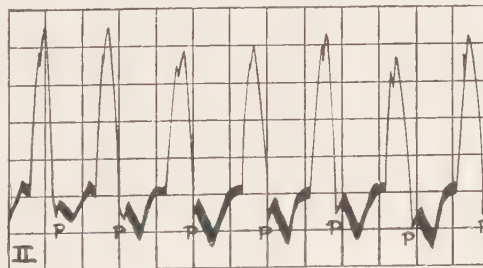


FIG. 193.—Record in lead II showing ventricular tachycardia with retrograde conduction. Note that each P wave immediately succeeds the QRS complexes. The QRS complexes are of high amplitude, of bizarre contour, and show variations in the form of the complexes. Record obtained from a man, aged fifty-three years, with coronary sclerosis.**

invariably displays some degree of irregularity. Retrograde conduction has been observed in ventricular tachycardia, as is shown in Figure 193. The abnormal complexes are usually of high amplitude; however, exceptions to this occur (Fig. 189).

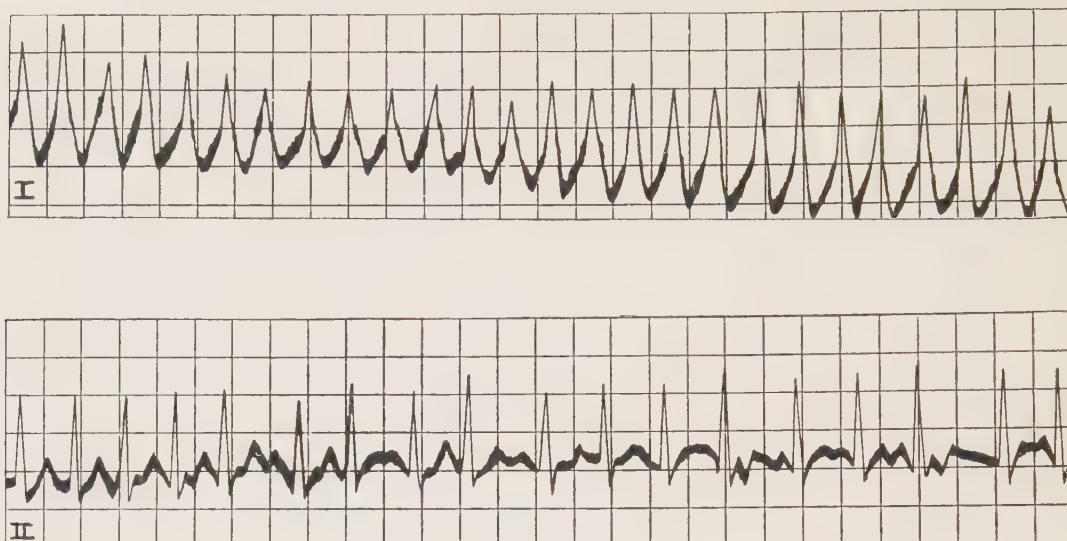


FIG. 194.—Records in leads I and II of one patient, taken just a few minutes apart, showing ventricular tachycardia and auricular flutter. Record obtained from a man aged forty-nine years.**

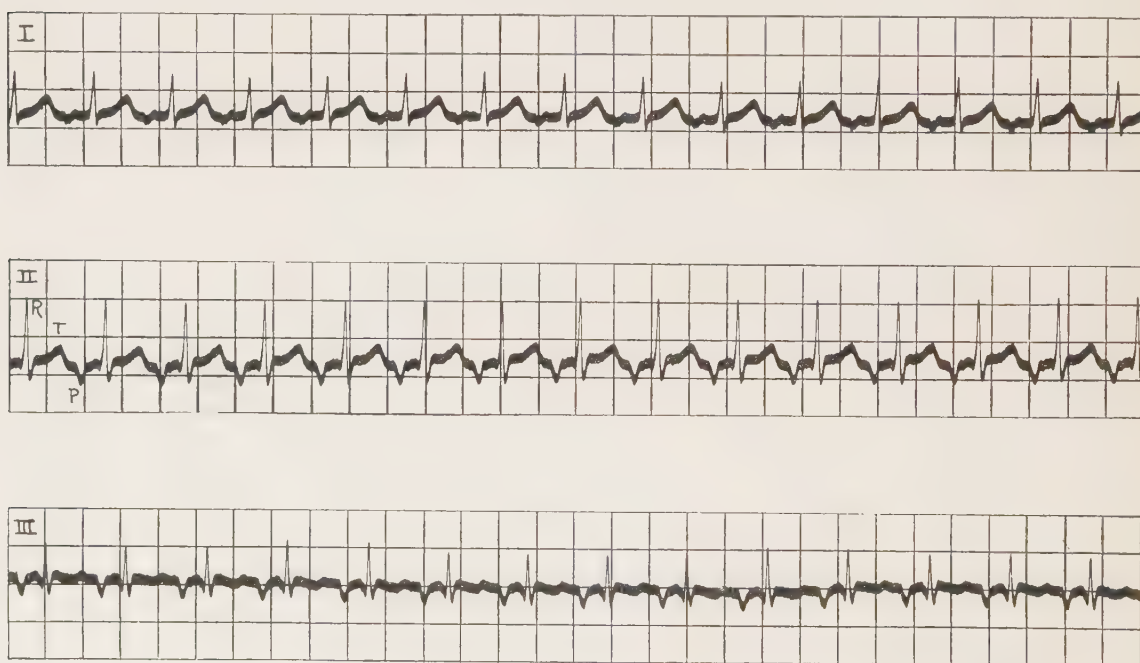


FIG. 195.—Record showing a paroxysm of auricular tachycardia. The P waves in all leads are inverted. The rate was 143 contractions each minute. Record obtained from a woman, aged fifty-seven years, with essential hypertension.

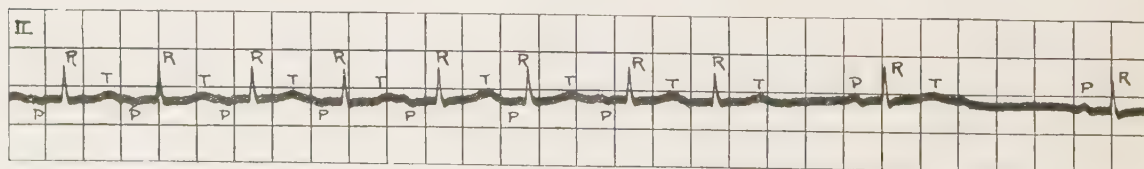


FIG. 196.—Record in lead II showing a period of auricular tachycardia and its termination. The P waves during the period of acceleration are inverted. Record obtained from a man, aged eighty-three years, with coronary sclerosis.

Ventricular tachycardia at times is associated with other abnormalities. It has been observed together with both auricular fibrillation and auricular tachycardia and, in Figure 194, is shown its coëxistence with auricular flutter.

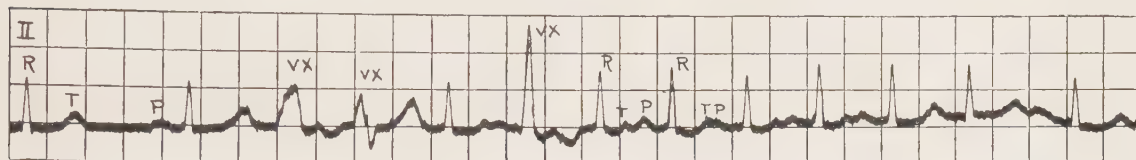
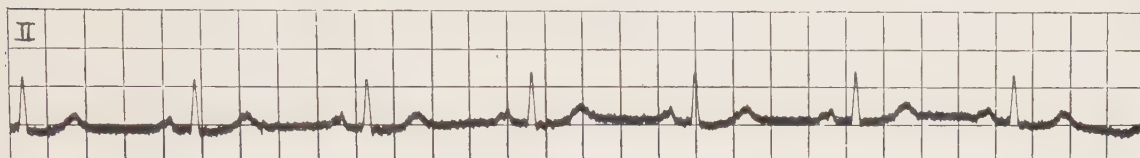


FIG. 197.—Record in lead II showing the inception of a short period of tachycardia which had its origin at, or near, the sino-auricular node. Note the premature contractions of varying forms which precede the period of acceleration. Record obtained from a man, aged sixty-two years, with coronary sclerosis.

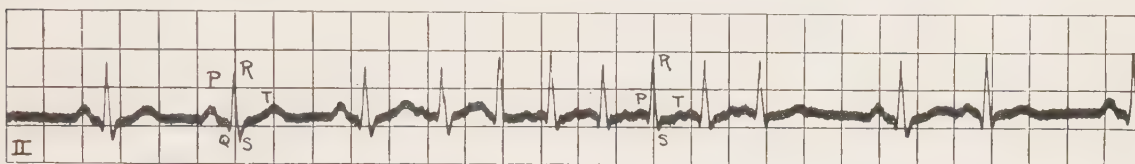


FIG. 198.—Record in lead II showing a short paroxysm of nodal (A-V) tachycardia. Note the diminution, to 0.08 second, of the P-R intervals. The rate during the paroxysm is 214. Record obtained from a man, aged seventy-six years, with coronary disease.

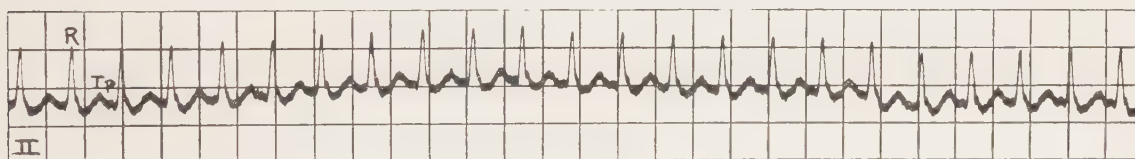


FIG. 199.—Record in lead II showing nodal (A-V) tachycardia. The P-R interval is markedly diminished to only 0.04 second. Record obtained from a woman, aged thirty-six years, with hyperfunctioning adenomatous goiter.

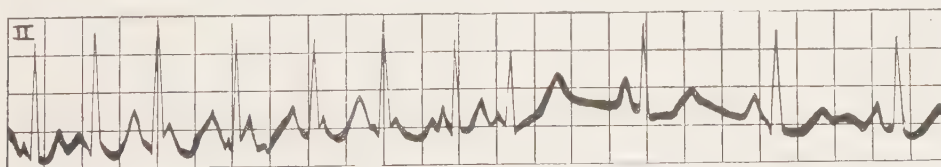


FIG. 200.—Record in lead II of a paroxysm of tachycardia, showing its termination. The paroxysm apparently arose in the junctional tissues. Record obtained from a woman, aged fifty-seven years, with coronary sclerosis.**

The presence of ventricular tachycardia is always significant. I have never observed it when unmistakable evidence of serious cardiac disease was lacking. It has been most often observed in coronary disease.

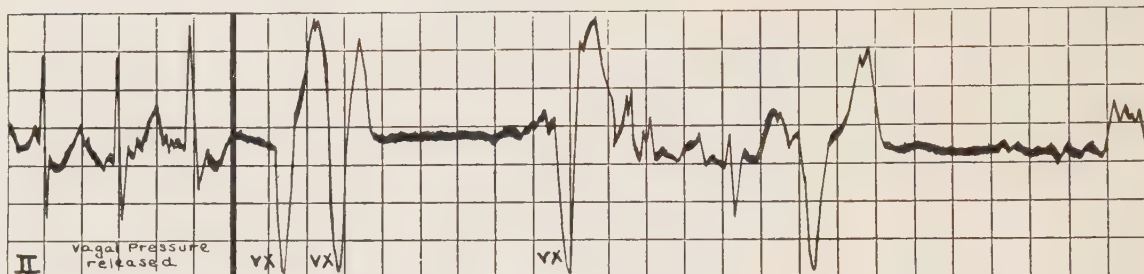
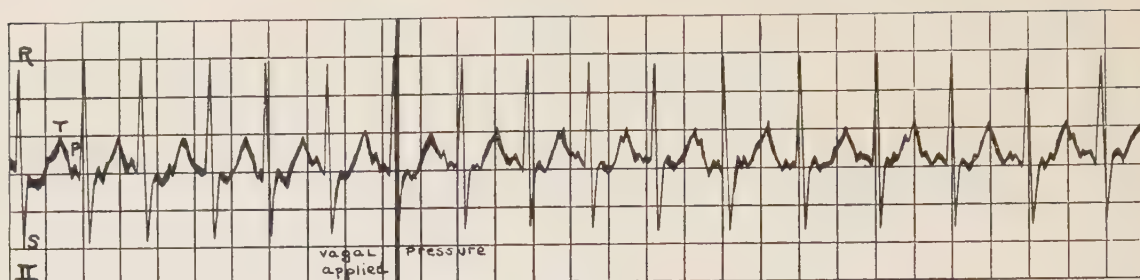


FIG. 201.—Records, continuous in lead II, showing a paroxysm of nodal tachycardia which terminates after the application of right vagal pressure. Note the marked diminution of the P-R intervals during the paroxysm. The abnormal rhythm is succeeded by premature contractions of high amplitude, ventricular in origin. Record obtained from a man, aged seventy years, with coronary sclerosis accompanied by angina pectoris and paroxysmal tachycardia.**

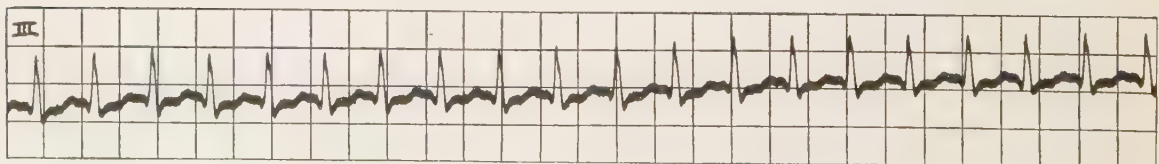
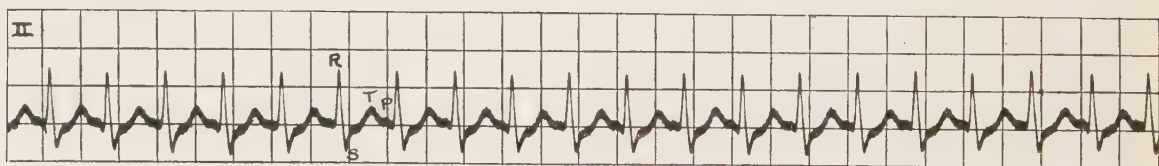
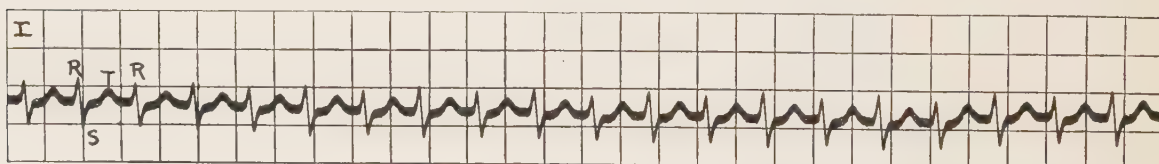


FIG. 202.—Record showing paroxysmal, nodal (A-V) tachycardia. Note the P waves of very low amplitude, and diminution in the P-R intervals (0.05 second). The rate is 195 contractions each minute. There is preponderance of the right ventricle. Record obtained from a woman, aged thirty-six years, without objective evidence of heart disease.

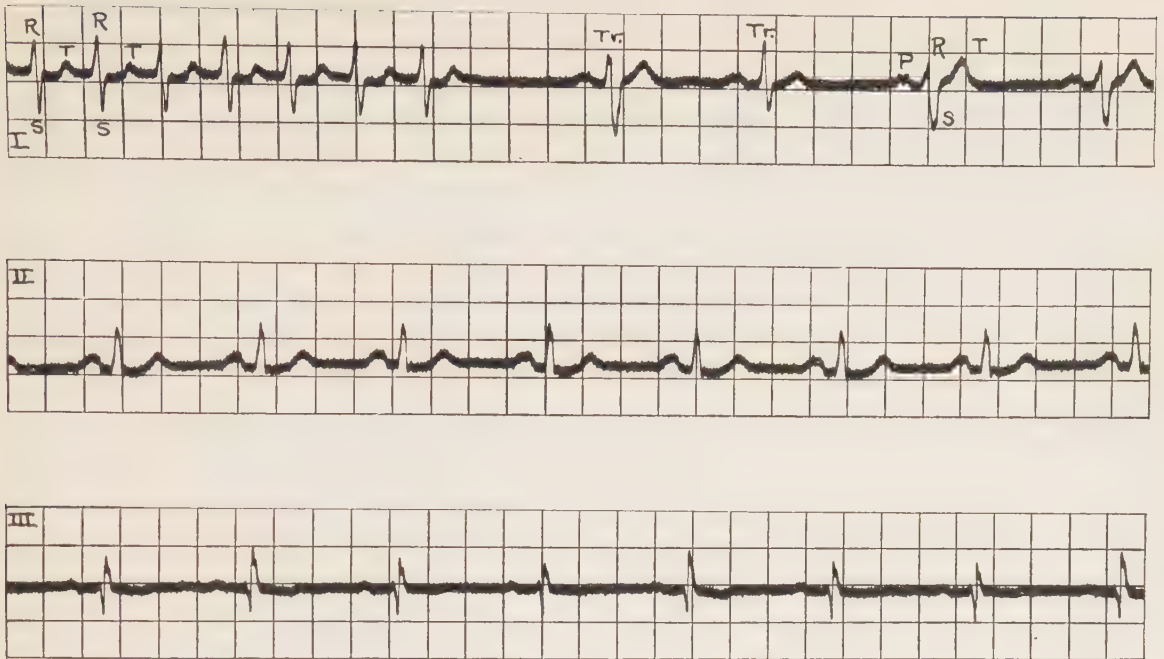


FIG. 203.—Record showing the termination of a paroxysm of nodal (A-V) tachycardia which occurs in lead I. Restoration of normal rhythm occurs after the appearance of two transitional complexes. Note the absence of the P waves during the paroxysm of tachycardia. Apparently they are submerged in the QRS complexes. The rate during the paroxysm is 205 contractions each minute. Record obtained from a man, aged forty-six years, with hypertensive heart disease.

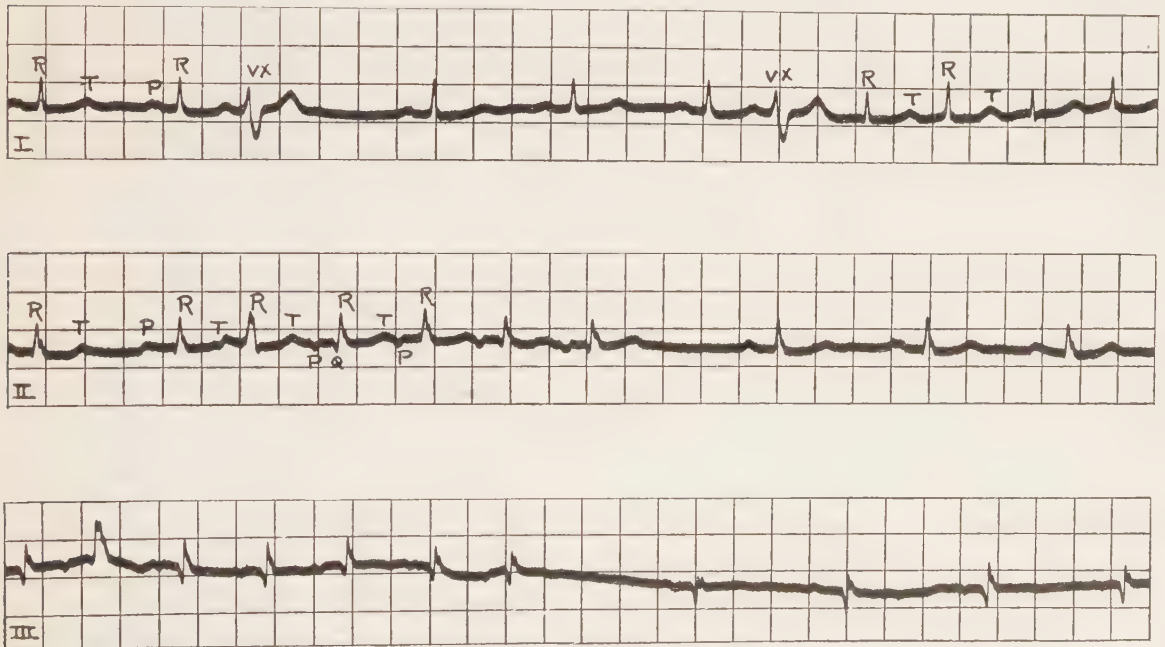


FIG. 204.—Record showing short paroxysms of nodal (A-V) and auricular tachycardia. Lead I shows normal rhythm interrupted by a premature contraction and followed by a short paroxysm of nodal (A-V) tachycardia. Note the absence of the P waves which apparently coincide with the R waves. In lead II another short paroxysm of tachycardia interrupts the normal rhythm; but here the P waves are present and inverted, indicating the point of origin in the auricle. Record obtained from an obese woman, aged fifty-nine years, with essential hypertension.

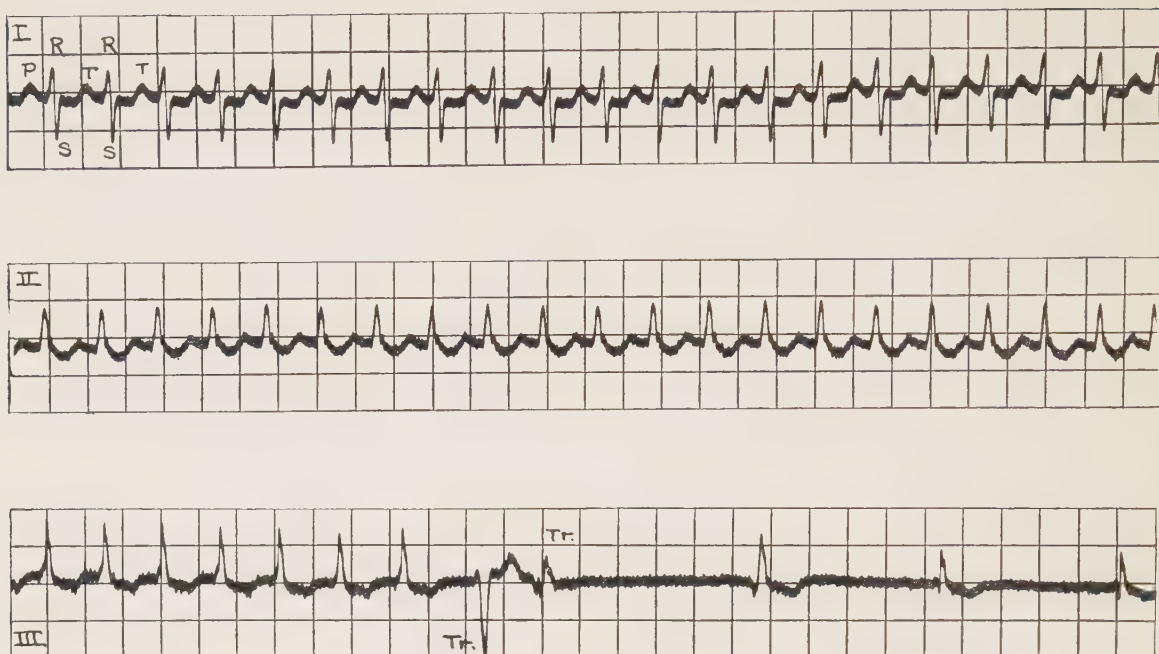


FIG. 205.—Record showing paroxysmal nodal tachycardia with termination of the attack in lead I. The P waves during the paroxysm are not visible for they coincide with and are submerged in the QRS complexes. The rate, which was 205 contractions each minute during the paroxysm, with normal rhythm became established at 77 contractions each minute. Two transitional complexes precede the establishment of normal rhythm. Record obtained from a man, aged fifty-four years, with hypertensive heart disease.

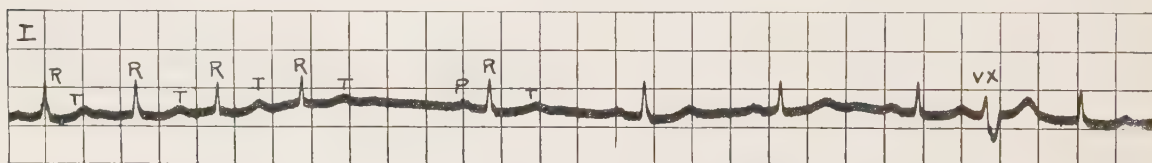


FIG. 206.—Record in lead I showing the termination of a paroxysm of nodal (A-V) tachycardia. Note the absence of the P waves in the complexes during the tachycardia. A premature contraction, apparently of ventricular origin, interrupts the restored normal rhythm. Record obtained from a woman, aged fifty years, with essential hypertension.

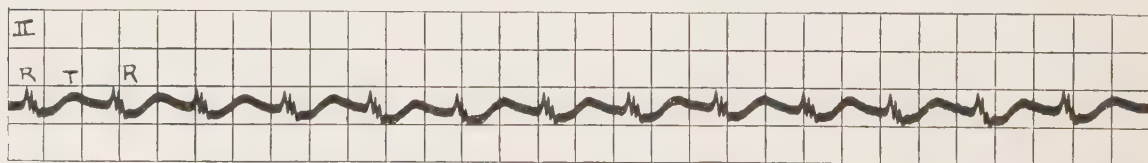


FIG. 207.—Record in lead II showing nodal (A-V) tachycardia. Note the absence of the P waves. The QRS complexes are notched. Record obtained from a man, aged sixty-eight years, with coronary sclerosis.

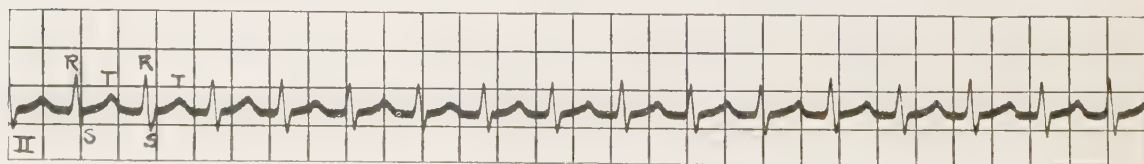


FIG. 208.—Record in lead II showing nodal (A-V) tachycardia. Note the absence of the P waves. Record obtained from a man, aged thirty-nine years, with attacks of paroxysmal tachycardia.

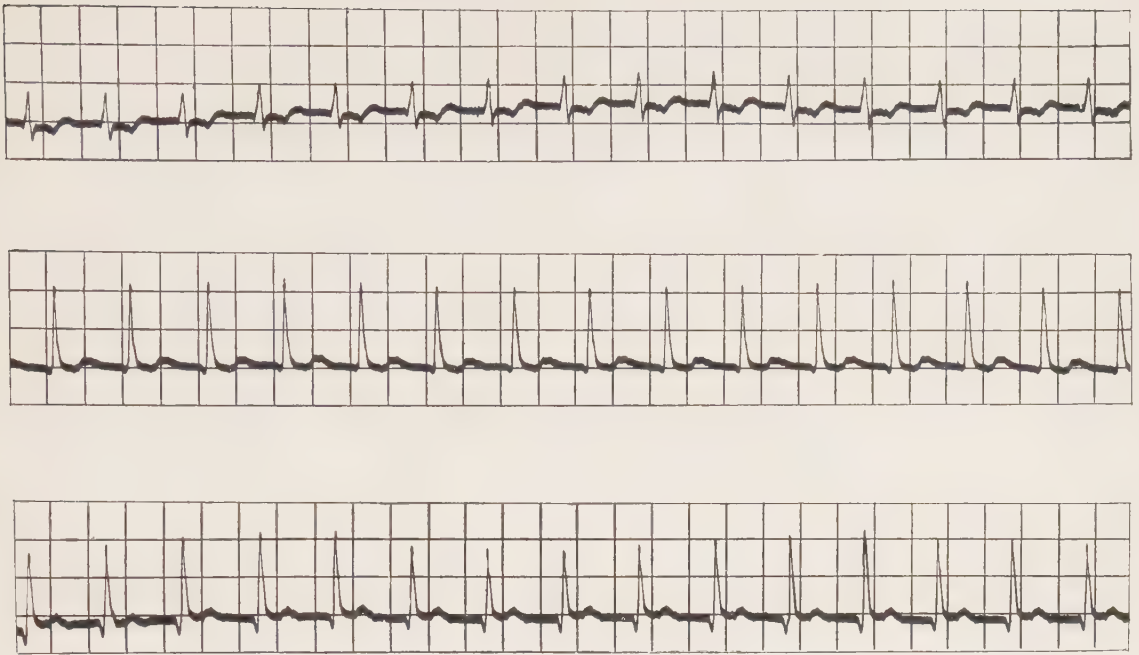


FIG. 209.—Record in all leads showing nodal (A-V) tachycardia. Record obtained from a child, aged seven years, with attacks of convulsive syncope attending the extremely rapid paroxysms of cardiac acceleration. The succeeding three records are from the same patient and were obtained within a period of several days.

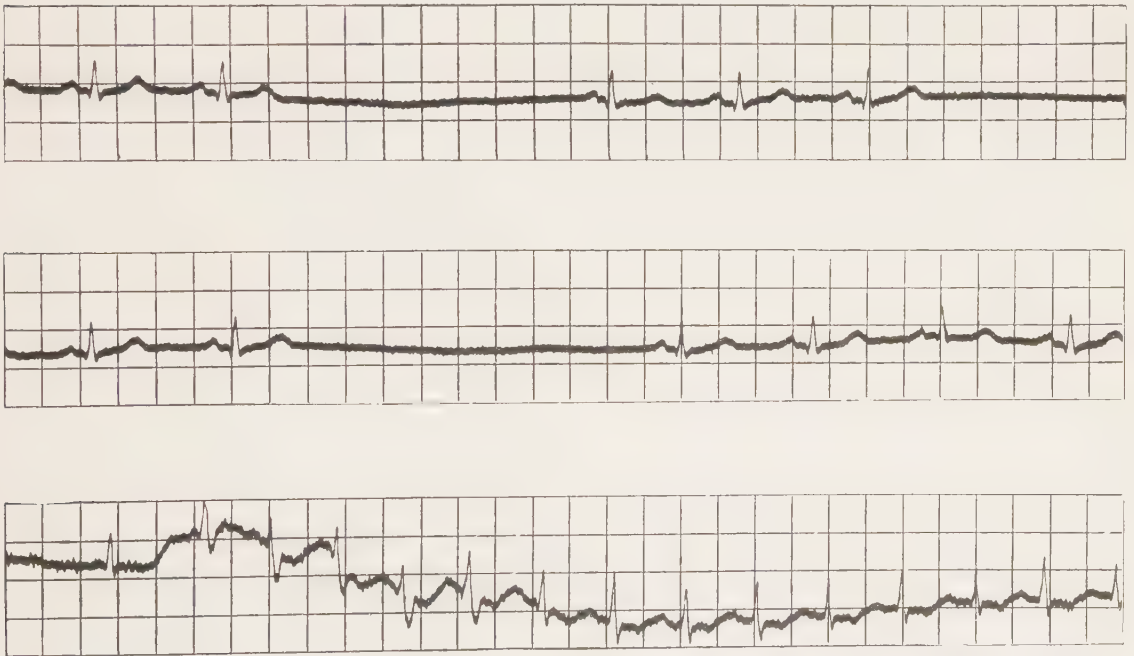


FIG. 210.—Record of the same patient (Fig. 209) taken in lead I. It shows long periods of cardiac asystole and periods of nodal (A-V) tachycardia.

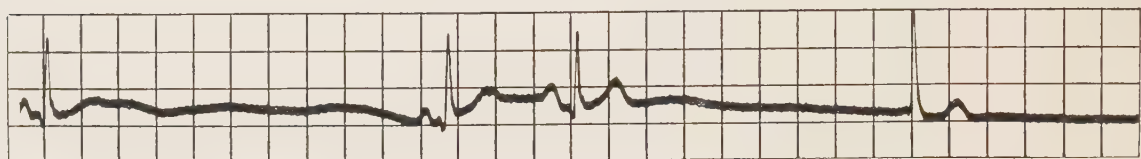
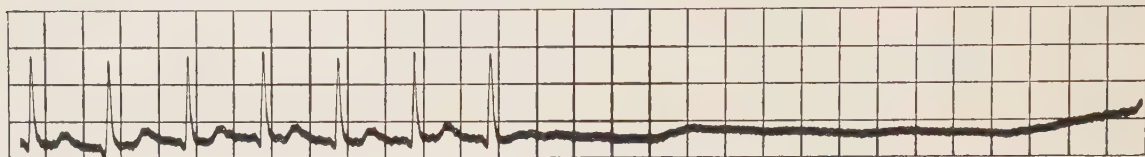
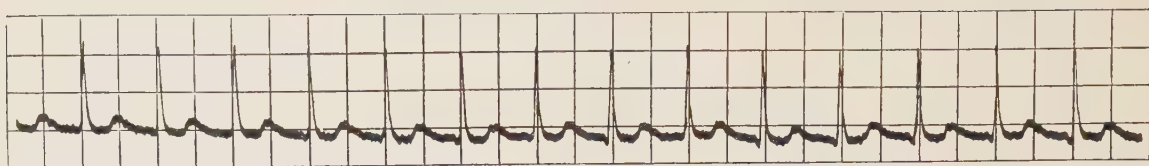


FIG. 211.—Record of the same patient (Fig. 210), taken in lead II, showing changes similar to those seen in the preceding figure.

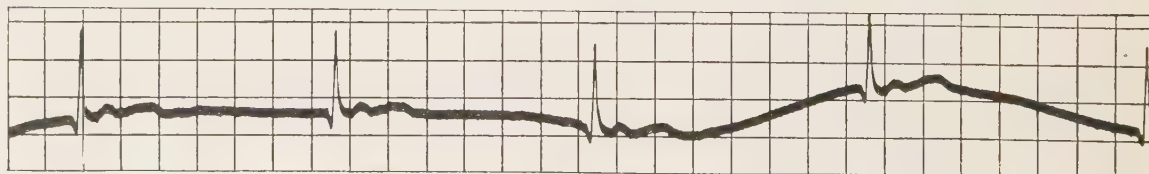
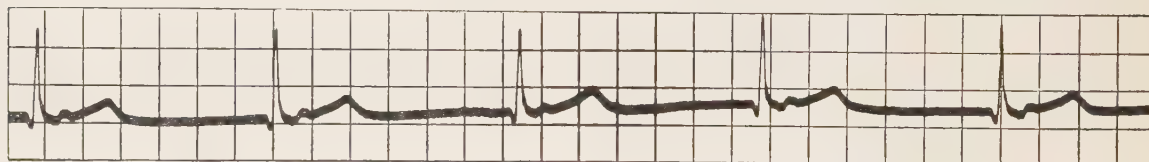
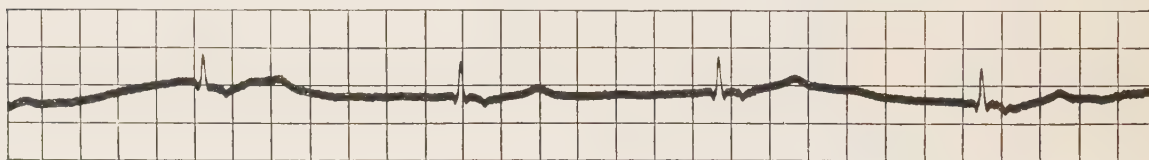


FIG. 212.—Record of the same patient (Fig. 211), in all leads, showing a period of slow nodal (A-V) rhythm with retrograde conduction.

AURICULAR TACHYCARDIA

In auricular tachycardia the area in which the ectopic impulse is formed is at some portion in the auricular musculature away from the sino-auricular node. Each auricular

beat is followed by a ventricular beat, and the rate does not exceed 200 each minute. The inversion of the P waves (negative P waves) in all leads indicates the ectopic origin of the tachycardia (Figs. 195 and 196). The ventricular components of the electrocardiogram remain unaltered, for the ventricle responds to supraventricular impulses that have traversed normal paths beyond their point of origin. In Figure 197 a paroxysm of tachycardia is shown in which the complexes appear to be normal; this indicates, presumably, that the paroxysm arose in the immediate vicinity of the sino-auricular node.

Auricular tachycardia occurs in the presence of heart disease; yet it has been observed occasionally in cases in which evidence of injury to the heart has not been demonstrable. The prognosis is more favorable than in the ventricular type.

NODAL (A-V) TACHYCARDIA

Nodal (A-V) tachycardia has its origin in a focus of abnormal irritability in the auriculoventricular node. It has been produced experimentally by depression of the sino-auricular node and by direct stimulation of the auriculoventricular node. The electrocardiograms, as in the other forms of ectopic tachycardia, display series of successive premature con-

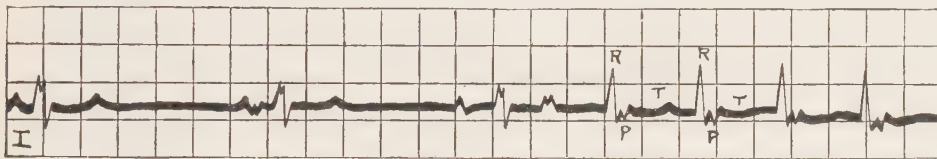


FIG. 213.—Record in lead I showing the onset of nodal (A-V) tachycardia with retrograde conduction. Note the inverted P waves just following the QRS complexes. Record obtained from a woman, aged forty-nine years, with essential hypertension.**

tractions. Both in nodal (A-V) premature contractions and in nodal (A-V) tachycardia three types are recognized. Presumably the type of nodal tachycardia depends on the site in the node of the origin of the impulse.

1. The P-R interval may be reduced; this probably occurs when the impulse arises in the upper portion of the node. Such records are shown in Figures 198 to 202.

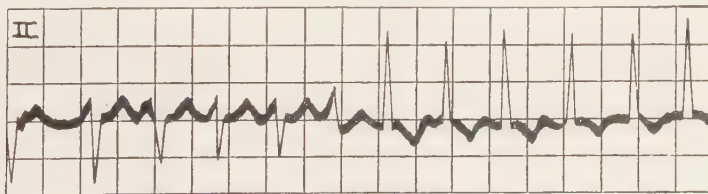


FIG. 214.—Record in lead II showing a paroxysm of tachycardia with sudden reversal of mechanism. Record obtained from a man, aged thirty-eight years, with slight cardiac hypertrophy and frequent paroxysms of tachycardia. In this case are exhibited some phases that arise apparently in the auricle and others that presumably arise in the ventricle.**

2. The P wave may be absent, apparently coinciding with the QRS complex and at times increasing its amplitude. This type is presumed to result when the ectopic focus occupies the central portion of the node and the contraction of the auricles and the ventricles is synchronous (Figs. 203 to 212).

3. The P wave may occur just after the R wave, or at times before its completion. Either of these occurrences gives rise to the so-called "R-P interval." The P wave, under these conditions, is invariably inverted (negative). Records of this sort supposedly indicate that the abnormal focus of irritability is situated in the lower portion of the node and that contraction of the ventricles precedes that of the auricles (Fig. 213). Occasionally nodal (A-V) and auricular tachycardia coexist in a record, as displayed in Figure 204. The arrest of a paroxysm of nodal (A-V) tachycardia following vagal pressure is seen in Figure 201.

In Figure 214 is shown a rather unusual record which exhibits reversal of the waves and presumably is a record of nodal (A-V) tachycardia.

Nodal (A-V) tachycardia is frequently observed when examination of the heart fails to disclose evidence to justify the diagnosis of heart disease. Nevertheless, this form of tachycardia has been found to occur in the presence of well marked, serious disease. The prognosis depends on the type and the degree of the associated cardiopathy.

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Chapter XII

NODAL (A-V) RHYTHM AND VENTRICULAR ESCAPE

NODAL (A-V) RHYTHM

Automatism of the auriculoventricular node was first demonstrated by Engelmann following the application of the first Stannius ligature in the frog and the function thus assumed by the node has been termed "nodal rhythm." It has been produced experimentally by vagal stimulation, by the depression of the sino-auricular node through cooling, by actual destruction, and under conditions of asphyxia.

This abnormal rhythm is usually slow, and changes in the waves of the electrocardiogram occur identical with the altered complexes of nodal premature contractions and nodal

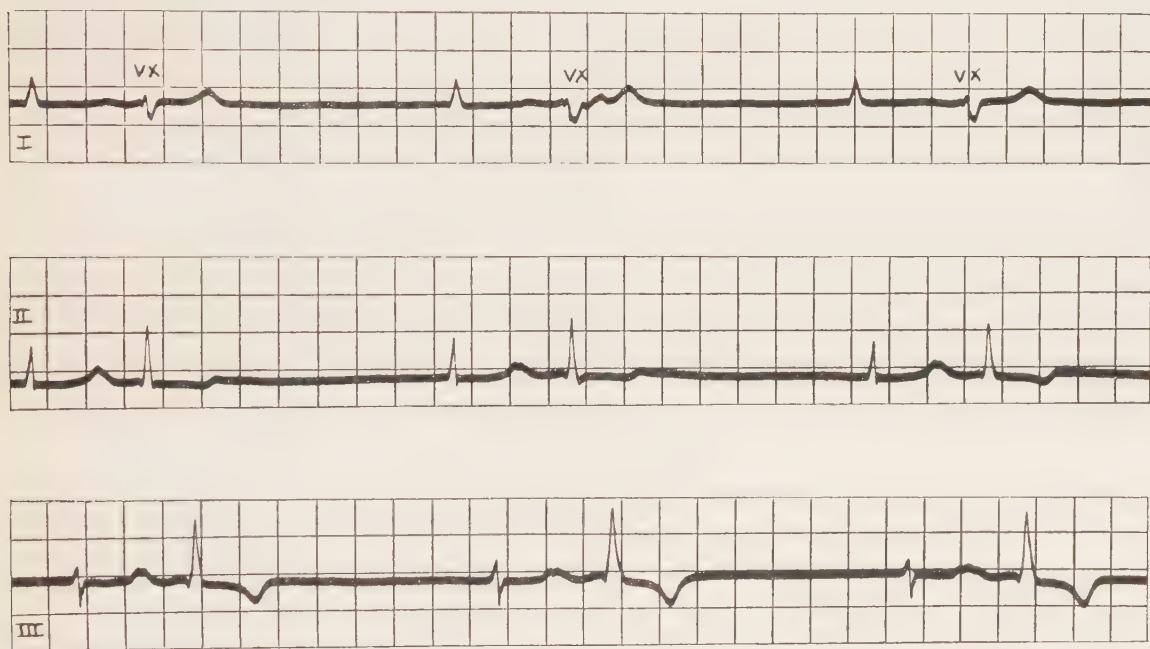


FIG. 215.—Record showing nodal (A-V) rhythm with alternating premature ventricular contractions. Note in the dominant rhythm the absence of the P wave which apparently occurs coincidentally with the R wave. Slight preponderance of the left ventricle is evident. Record obtained from a woman, aged thirty-one years, without evidence of organic heart disease.

tachycardia. Three types of nodal rhythm are recognized. The P-R interval may be diminished: in such a case the area of production of the abnormal stimulus may be assumed to be situated in the upper portion of the node, and the auricles then contract just in advance of the ventricles. The P wave may not be visible in the record: then, the assumption is that the P wave occurred at the time of development of the QRS complex, and presumably simultaneous contraction of auricles and ventricles was excited from an ectopic focus in

the central portion of the node. The P wave may follow the QRS complex: usually under such conditions it occurs just before the beginning of the T wave and apparently it is due to an abnormal focus in the lower portion of the node. This sequence is accepted as evi-

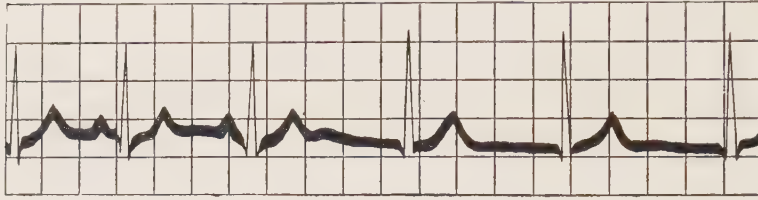


FIG. 216.—Record in lead II showing the inception of nodal (A-V) rhythm. Note the absence of the P waves in the last three complexes and the increased amplitude of the R waves. Record obtained from a woman, aged thirty-three years, without evidence of organic heart disease.**

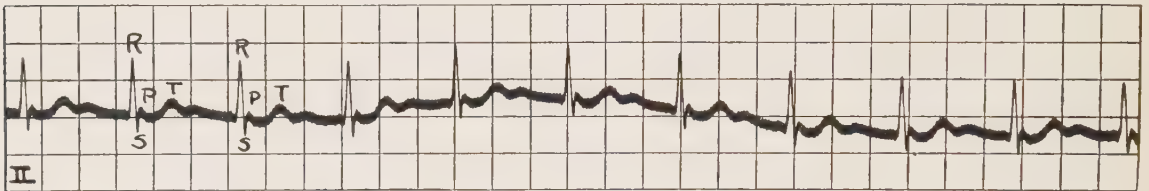


FIG. 217.—Record in lead II showing nodal (A-V) rhythm with retrograde conduction. Record obtained from a man, aged sixty-five years, with hypertensive heart disease and severe secondary anemia.

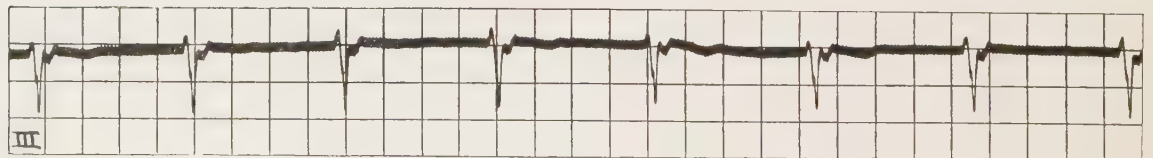
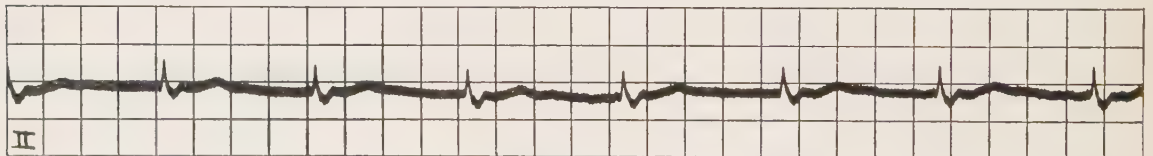
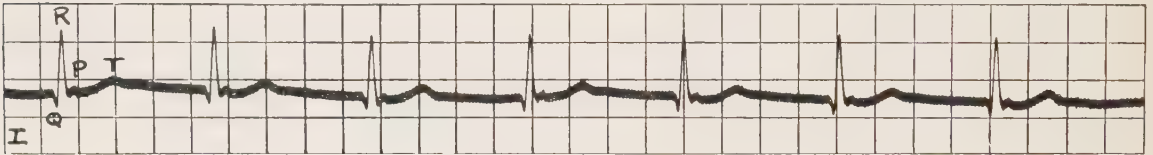


FIG. 218.—Record showing nodal (A-V) rhythm with retrograde conduction. Record obtained from a man, aged forty-nine years, with chronic morphinism.

dence that the ventricles contracted in advance of the auricles and that the latter were activated by retrograde conduction. Examples of nodal (A-V) rhythm are shown in Figures 215 to 218.

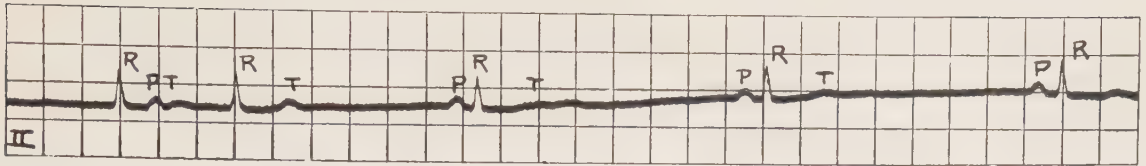


FIG. 219.—Record in lead II showing a migratory pacemaker. Note the differences in the intervals between the P and the R waves. Record obtained from a man, aged fifty-four years, without evidence of organic heart disease.

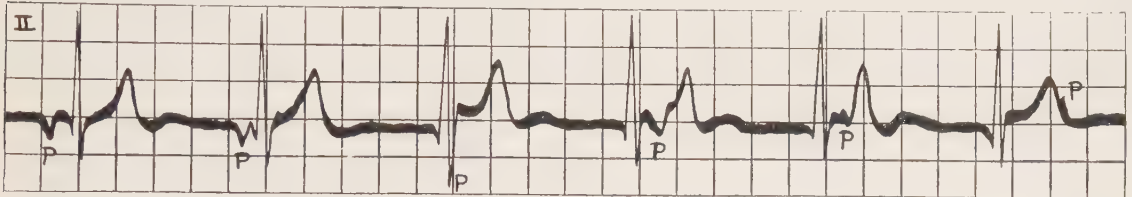


FIG. 220.—Record in lead II showing a migratory cardiac pacemaker. Note that the relationship of the inverted P waves to the ventricular rhythm is varying continually. This results in the ventricular rate being faster than that of the auricles. Record obtained from a man, aged twenty years, with congenital heart disease.**

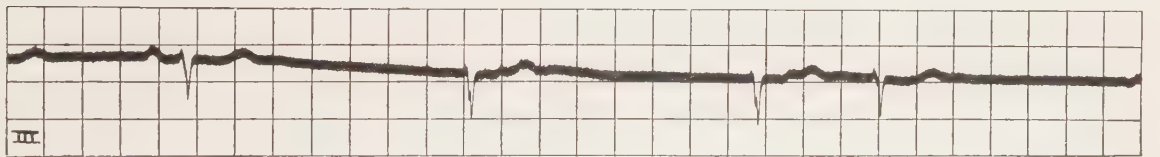
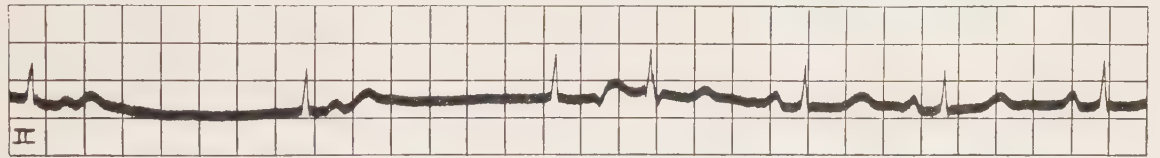
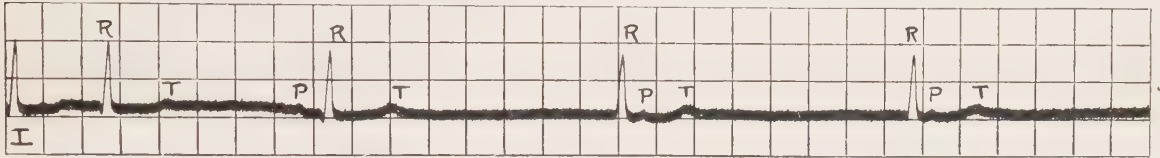


FIG. 221.—Record showing ventricular escape with temporary restoration of normal rhythm in lead II. The ventricular frequency during the periods of escape is 46 contractions each minute. A moderate degree of preponderance of the left ventricle is present. Record obtained from a woman, aged fifty-one years, without demonstrable evidence of organic heart disease.

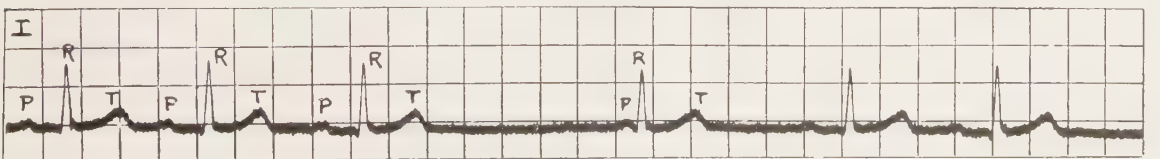


FIG. 222.—Record in lead I showing ventricular escape and slight delay in auriculoventricular conduction; the P-R interval is 0.24 second. Record obtained from a man, aged thirty-six years, without evidence of organic heart disease. The delayed auriculoventricular conduction was subsequently abolished by means of atropine.

Occasionally electrocardiograms are observed from which it can be assumed that the area of origin of the impulse varied from cycle to cycle and ranged from its normal site in the sino-auricular node to various levels in the auriculoventricular node. These records often display a constantly changing relationship of the P waves to the QRS complexes. The term "migratory pacemaker" has been applied to this changing area of origin of the

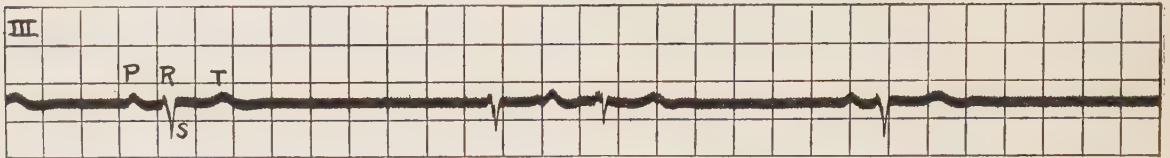


FIG. 223.—Record in lead III showing ventricular escape. The complex which succeeds recovery is apparently a beat which arises in the junctional tissues. Record obtained from a woman, aged forty-one years, without evidence of organic heart disease.

impulse. Records produced under the conditions imposed by such a pacemaker are shown in Figures 219 to 221.

Nodal rhythm is observed in apparently normal hearts and may be the result of purely neurogenic influences which affect the vagi. Its occurrence in heart disease is not unusual, but it is not characteristic of any cardiopathy. It is a constant finding when death is

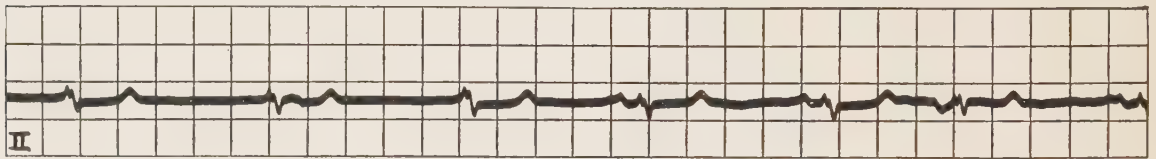


FIG. 224.—Record in lead II showing ventricular escape. Record obtained from a woman, aged forty-six years, who had chronic rheumatic endocarditis with mitral stenosis and insufficiency.

taking place and uniformly it ushers in the prelethal bradycardia. This phenomenon will be more fully discussed in a later chapter.

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VENTRICULAR ESCAPE

The ventricles occasionally escape from the control of the sino-auricular node because of the development of autonomy of the auriculoventricular node. Two forms of ventricular escape have been described: one results from depression of the sino-auricular node; the other, from excitation of the auriculoventricular node. The latter form occurs infrequently. Recognition of the two forms depends largely on the rate of the sino-auricular pacemaker from which the ventricles escape and on the rate of the ventricular pacemaker which escapes. The ventricular complexes remain unaltered, owing to their activation through supraventricular paths.

The differentiation of ventricular escape and nodal (A-V) rhythm is made only by the electrocardiogram. In ventricular escape, the heart is under the control of two pacemakers: in nodal (A-V) rhythm, only the auriculoventricular node controls the heart. Occasionally the rate of the ventricles exceeds that of the auricles. Records displaying ventricular escape are shown in Figures 222 to 224.

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Chapter XIII

INDIVIDUAL WAVE CHANGES

The individual waves of the electrocardiogram are subject to various influences which alter their contour, amplitude and direction. These alterations vary in significance depending on their character, on the waves involved, and on whether the waves in one lead or in all leads are affected.

INCREASED AMPLITUDE OF THE P WAVE

The P wave is frequently subject to changes, even in normal hearts. As previously stated, the P wave is usually rather low; the amplitude normally varies from 2 to 4 millivolts.

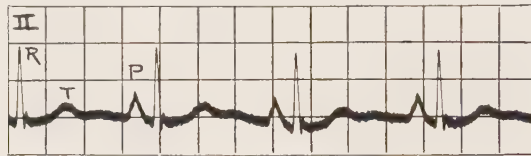


FIG. 225.—Record in lead II showing rather marked exaggeration of the amplitude of the P waves. Note extremely peaked character of the waves. Record obtained from a man, aged fifty-two years, with marked pulmonary emphysema.

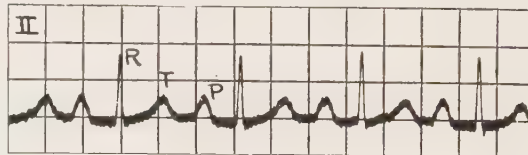


FIG. 226.—Record in lead II showing exaggeration in the amplitude of the P waves. Record obtained from a woman, aged forty-eight years, with hyperfunctioning adenomatous goiter.

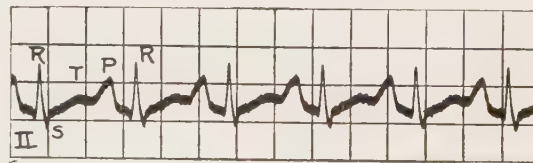


FIG. 227.—Record in lead II showing the P waves definitely increased in amplitude. Record obtained from a man, aged fifty-seven years, who had chronic rheumatic endocarditis with mitral stenosis.

volts, and the apex is rounded and is directed upward. At times, the amplitude is increased to 10 millivolts and the waves rise abruptly to pointed apices. Such complexes are frequently observed in cases of pronounced mitral stenoses and have led some observers to believe that they indicate auricular hypertrophy. However, the fact that the identical type of complex at times is observed in records from normal hearts throws doubt on this interpretation. Similar exaggerated, peaked P waves are observed in records which dis-

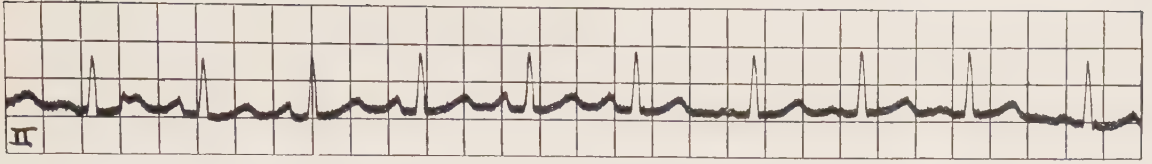


FIG. 228.—Record in lead II showing sudden diminution in the amplitude of the P waves. Record obtained from a woman, aged sixty-seven years, with hyperfunctioning adenomatous goiter.

play nodal (A-V) rhythm and tachycardia when the origin of the rhythm is ectopic (Figs. 225 to 227). A sudden diminution in the amplitude of the P waves is shown in Figure 228.

NOTCHING OF THE P WAVE

Distinct notching of the P wave frequently is observed. Usually the notching involves the apex or the descending limb of the wave. Lead II and leads II and III most frequently



FIG. 229.—Record showing notching and exaggeration of the P waves. Records from three patients.

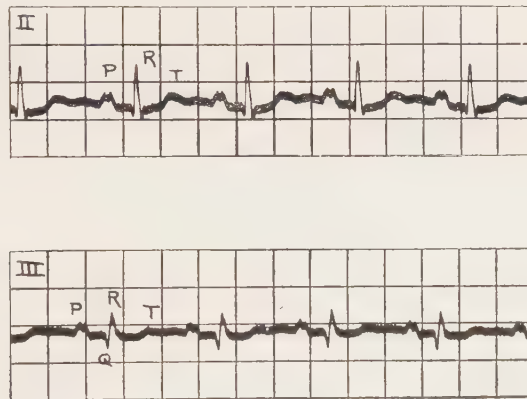


FIG. 230.—Record in leads II and III showing notching of the P waves. Record obtained from a man, aged thirty-five years, with essential hypertension.

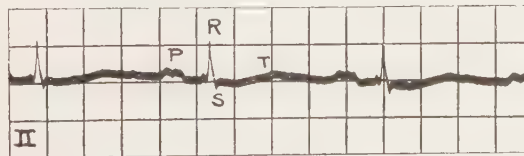


FIG. 231.—Record in lead II showing flattened and notched P waves. Record obtained from a man, aged forty-seven years, with exophthalmic goiter.

display this alteration in the contour of the wave, but at times all the leads are affected. The association of notching with increased amplitude of deflection is a common occurrence

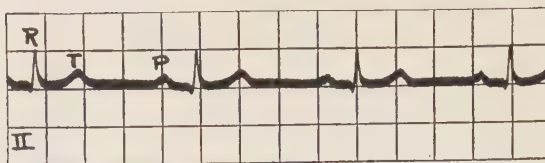


FIG. 232.—Record in lead II showing notching of low amplitude P waves. Record obtained from a man, aged seventy-seven years, with coronary sclerosis.

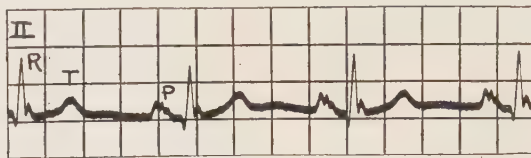


FIG. 233.—Record in lead II showing marked notching of the P waves. Record obtained from a man, aged sixty-seven years, with coronary sclerosis.

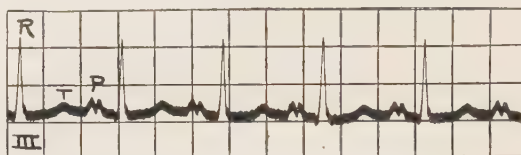


FIG. 234.—Record in lead III showing notching of the P waves. Record obtained from a man, aged seventy-six years, with aortic sclerosis.

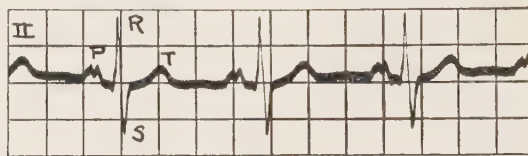


FIG. 235.—Record in lead II showing marked notching of the P waves. Record obtained from a man, aged forty-two years, without evidence of organic heart disease.

in mitral stenosis. In these cases the notching has been ascribed to auricular asynchronism; yet it occurs in normal hearts. The notching may be very slight or it may be so pronounced as to cause marked deformity of the wave (Figs. 229 to 235).

CHANGES IN THE DIRECTION OF THE P WAVE

Inversion (negativity) of the P wave in all leads has been shown to indicate the presence of an ectopic rhythm; that is, a rhythm having its origin at a site away from the sino-auricular node. The ectopic rhythm usually occurs at some point in the auricle; at times, in the auriculoventricular node.

Inverted or diphasic P waves, which occur only in lead III, are sometimes observed as transient phenomena. An attempt has been made to explain these phenomena by assuming that changes in muscle balance alter the relation of the pacemaker to the axis for lead III. The actual causes for these changes in direction of the P wave are not clear. Their occurrence in normal hearts has been observed repeatedly (Figs. 236 to 238).

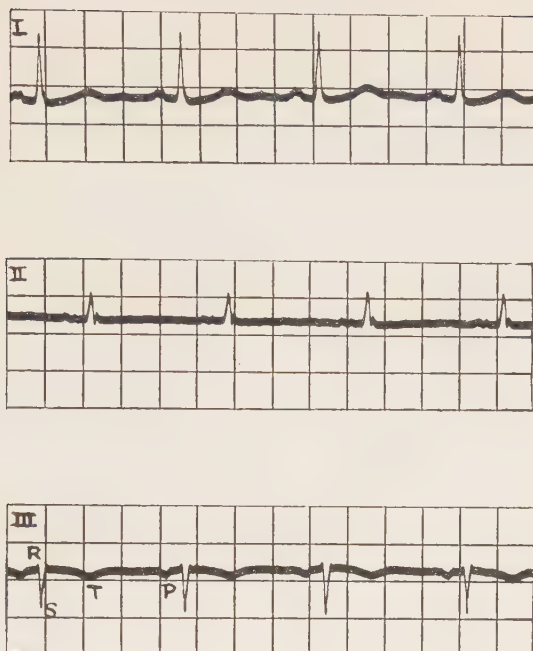


FIG. 236.—Record showing inversion of the P waves in lead III. Note the notching of the P waves in leads I and II. The T waves in lead II are inverted. Preponderance of the left ventricle is evident by the depression of the S waves in lead III. Record obtained from a woman, aged fifty-two years, with a moderate degree of essential hypertension.



FIG. 237.—Record in lead III showing inversion of the P waves. Record obtained from a man, aged seventy-seven years, with coronary sclerosis.

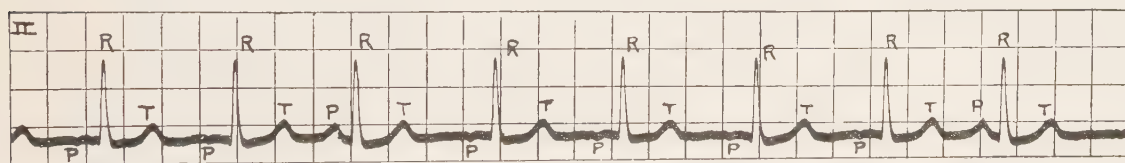


FIG. 238.—Record in lead II showing transient inversion of the P waves. Record obtained from a woman, aged fifty-three years, without evidence of heart disease.

CHANGES IN THE QRS COMPLEX

The QRS complex is often altered by changes in its contour which occur in isolated or combined leads. The normal QRS complex is very abrupt, peaked, and varying in amplitude from 10 to 15 millivolts or more, and the duration of the complex normally does not exceed 0.10 second.

In complexes of normal duration, the least noticeable change in composition is a thickening, or slurring, of the ascending or descending limb. This may occur in one lead or may

involve the complexes in all leads. Slurring may occur as a transient phenomenon in some records; in other records it may be a constant characteristic. This abnormality has little significance as an isolated phenomenon; but when its appearance is constant, in cases of organic heart disease, it may be the precursor of signs of greater disturbance in conduction.

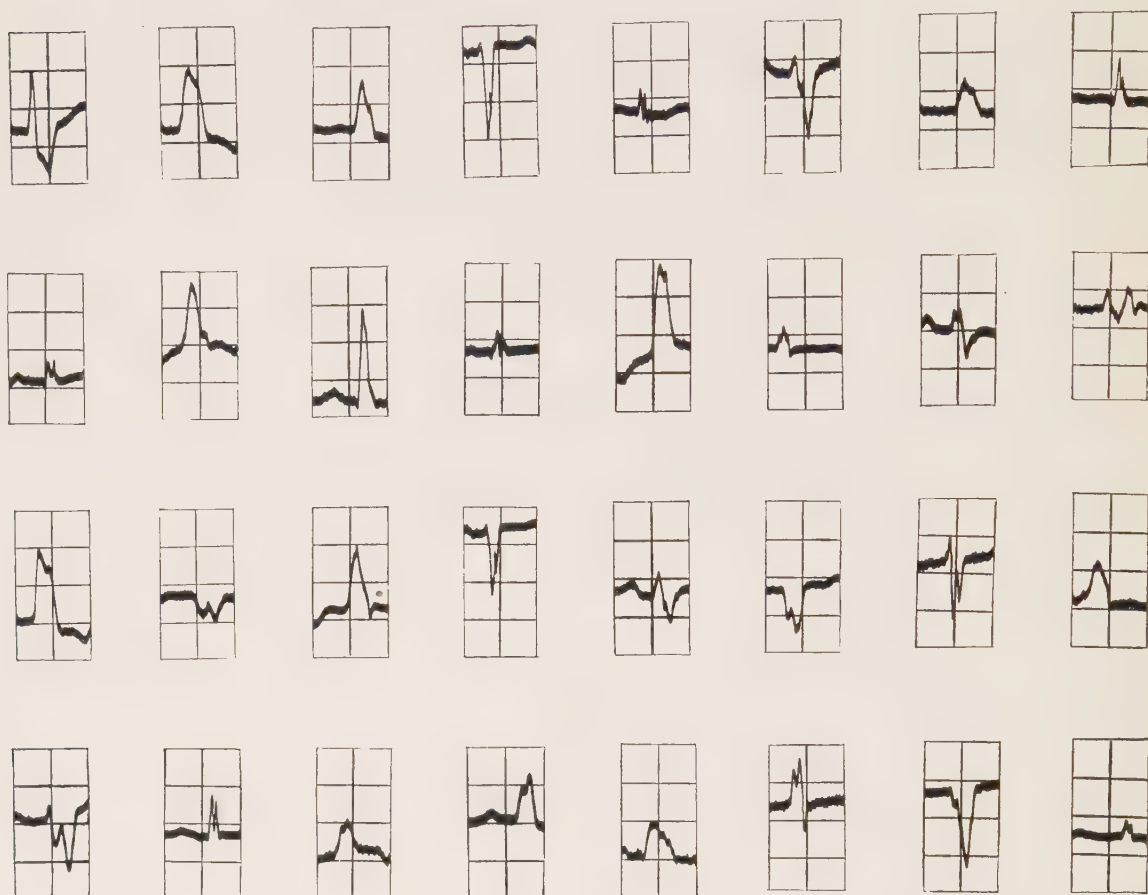


FIG. 239.—Records of thirty-two cases showing many variations in the contour of abnormal QRS complexes.

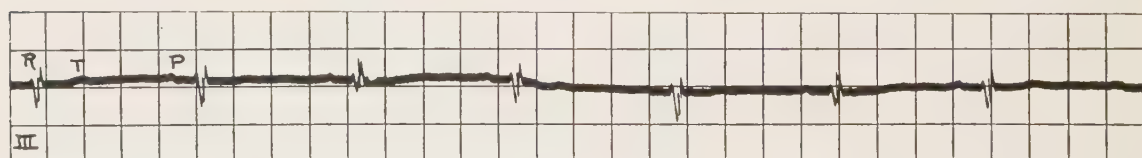


FIG. 240.—Record in lead III, showing continually changing direction of the QRS complexes which are deformed by well marked notching. Record obtained from a man, aged forty-six years, with essential hypertension.

Slight notching of the ascending limb, apex, or descending limb of the QRS complex frequently occurs temporarily in one or two leads. Caution must be exercised to avoid placing too much significance on such tracings, for numerous clinical records are now available which show the occurrence of such complexes in records of normal hearts.

Considerable significance is attached to definite notching of the QRS complexes, when it occurs in all leads, with intervals prolonged beyond the limits of normal. Records of

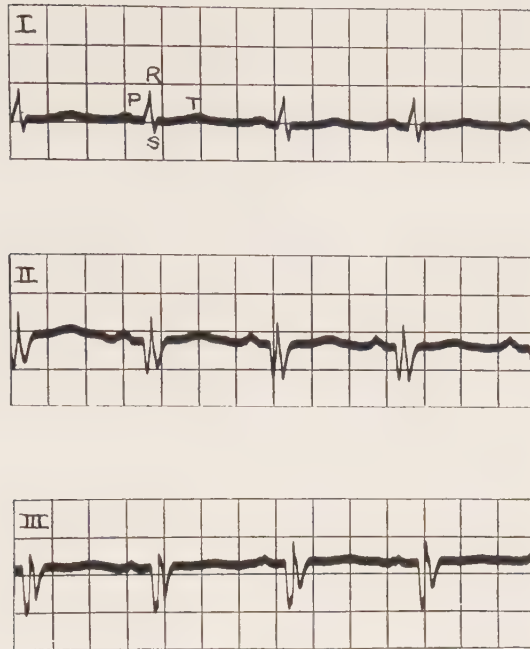


FIG. 241.—Record showing the so-called “W” notching of the QRS complexes in leads II and III. This is not a record of incomplete bundle-branch block. Record obtained from a man, aged forty years, with mild essential hypertension.

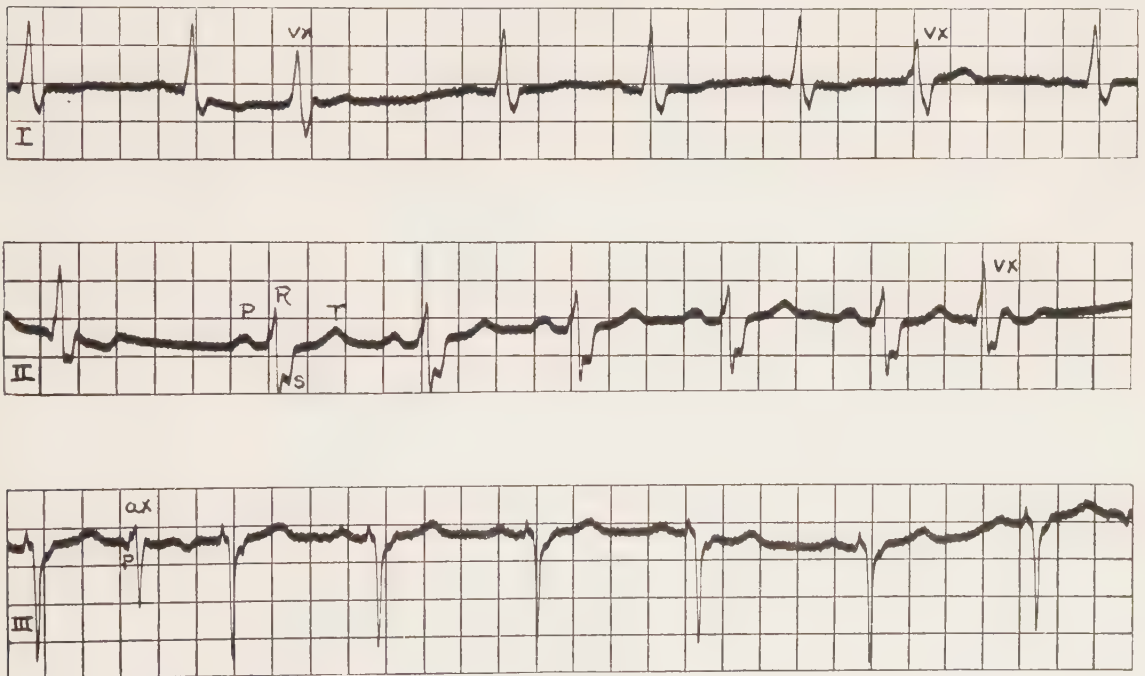


FIG. 242.—Record showing notching of the QRS complexes in lead II. Owing to the fact that fairly normal complexes comprise the dominant rhythm in leads I and III, this record does not embody the requirements of incomplete bundle-branch block. Note the peculiar notching of the QRS complexes in lead II. Premature contractions, from time to time, interrupt the rhythm; those in leads I and II arise in the ventricle, whereas that in lead III arises in the auricle. Marked preponderance of the left ventricle is present. The T waves in lead I are diphasic. Record obtained from a man, aged forty-two years, with hypertensive heart disease.

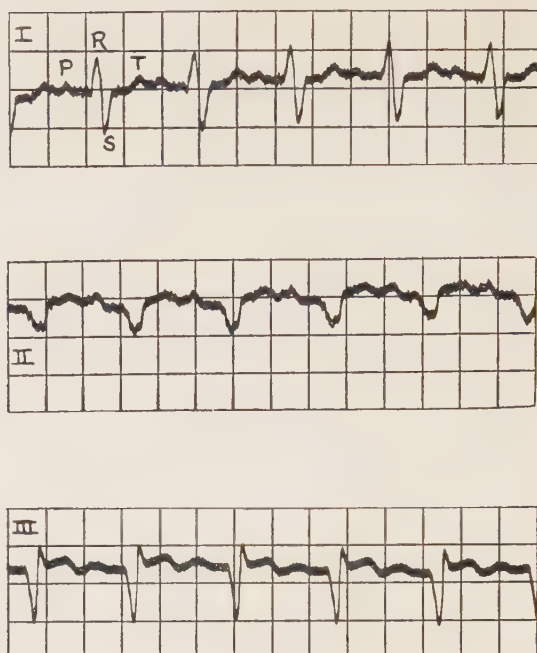


FIG. 243.—Record showing slight aberration of the QRS complexes in leads I and III and definite notching in lead II. Such a record is transitional between the type known as normal and the type characteristic of incomplete bundle-branch block. Record obtained from a man, aged sixty-four years, with hypertensive heart disease.

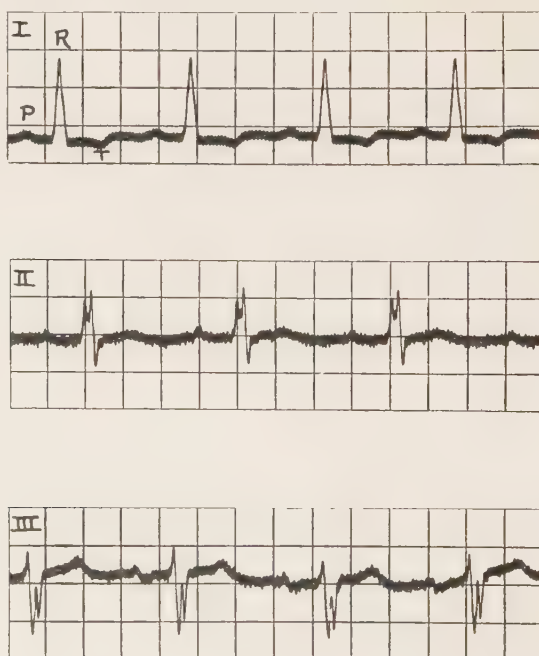


FIG. 244.—Record showing distinct notching of the QRS complexes in leads II and III. This does not represent an incomplete bundle-branch block. The T waves in lead I are inverted. Record obtained from a man, aged forty years, with a syphilitic aneurysm of the aortic arch.

this type are discussed fully under incomplete and complete bundle-branch block (Figs. 239 to 247).

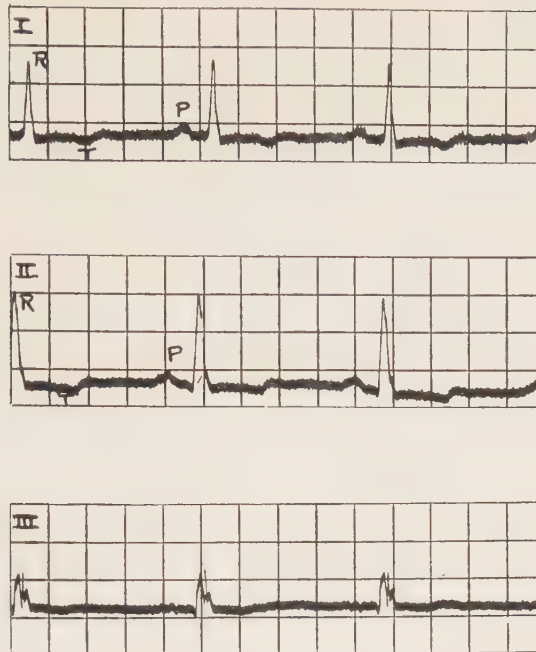


FIG. 245.—Record showing inversion of the T wave in leads I and II and extraordinary notching of the QRS complexes in lead III. Careful scrutiny reveals three distinct peaks in these complexes, with unusual uniformity of reproduction. Record obtained from a woman, aged fifty-six years, with essential hypertension which suggests a malignant type of the disorder.

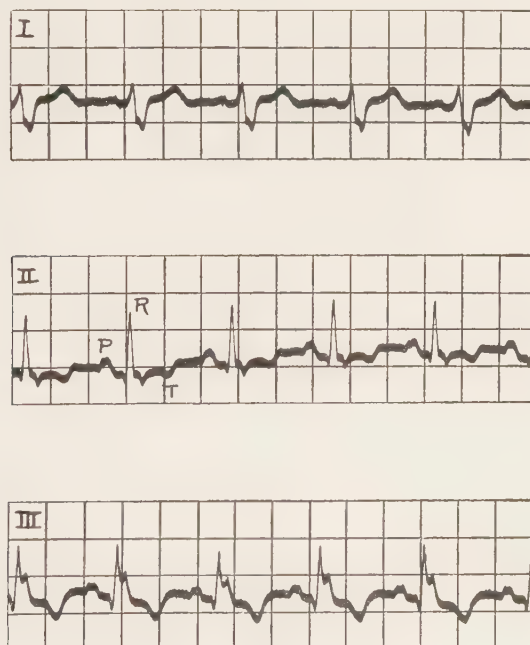


FIG. 246.—Record showing notching of the QRS complexes in leads I and III. The T waves in leads II and III are inverted. A record of this type does not meet the requirements of incomplete bundle-branch block. Record obtained from a woman, aged twenty-four years, without evidence of organic heart disease.



FIG. 247.—Record showing notching of the QRS complexes in leads II and III. The complexes in lead II show only slight notching of the ascending limbs, whereas in lead III the notching is well marked. A record such as this must not be confused with one of incomplete bundle-branch block. Record obtained from a man, aged sixty-five years, with no evidence of heart disease.

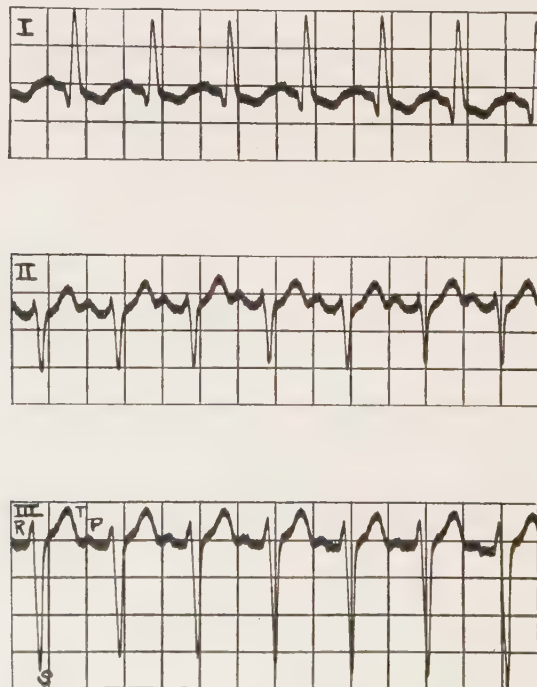


FIG. 248.—Record showing marked phasic variation in the amplitude of the S complexes in lead III. In addition to this finding, note the inversions of the T waves, in lead I and the marked preponderance of the left ventricle. Record obtained from a woman, aged thirty-one years, who had chronic rheumatic endocarditis with aortic insufficiency.

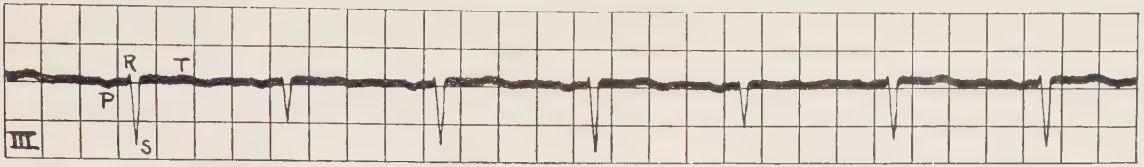


FIG. 249.—Record in lead III showing phasic variation in the amplitude of the QRS complexes. Note the inversion of the P waves. Record obtained from a woman, aged fifty-seven years, with coronary heart disease and angina pectoris.

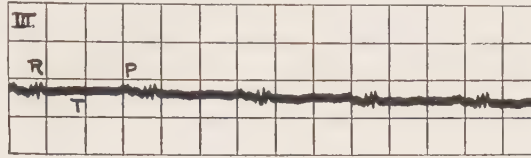


FIG. 250.—Record in lead III showing complexes of very low amplitude and triple notching of the QRS complexes. The T waves are inverted and the P waves are notched. Record obtained from an obese man aged fifty-two years.

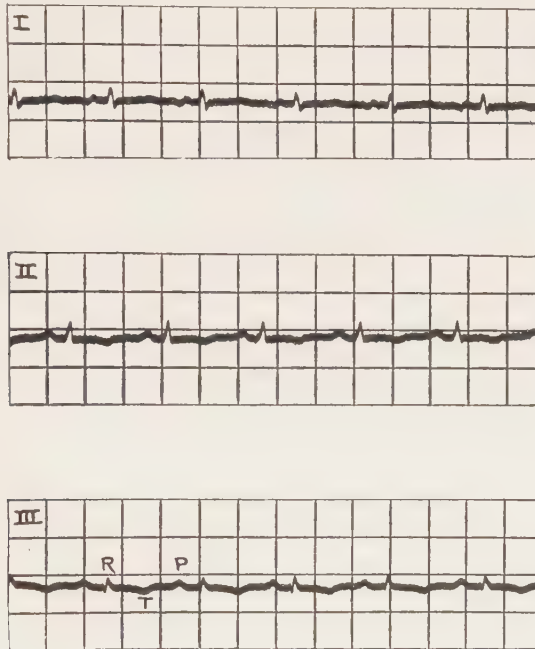


FIG. 251.—Record showing complexes of very low amplitude in all leads. Note the inversions of the T waves in leads II and III. Record obtained from a man, aged twenty-four years, with Pick's disease.

At times phasic or cyclic variations occur in the amplitude of the R waves in lead III. This is probably a respiratory effect from vagal influences (Figs. 248 and 249).

CHANGES IN THE T WAVE

Abnormalities in the T wave are frequent and some of them are of definite significance. As previously stated, the T wave is rounded and has an amplitude of 3 to 7 millivolts.

INCREASED AMPLITUDE OF THE T WAVE

At times a T wave of high amplitude is abrupt and peaked; at other times the blunt curved contour of the normal wave is maintained. Sometimes these waves of high amplitude are confined to lead II; however, they also are recorded in other leads. The exact nature of this phenomenon is not known; it frequently is observed in hyperthyroidism,

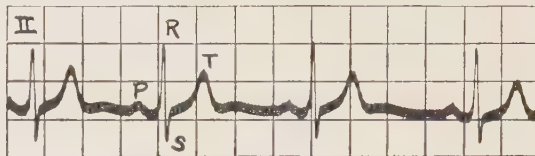


FIG. 252.—Record in lead II showing marked exaggeration in amplitude of the T waves. Record obtained from a man, aged twenty-two years, who had chronic rheumatic endocarditis with mitral stenosis and insufficiency.



FIG. 253.—Record in lead II showing exaggeration of the T waves. Record obtained from a woman, aged twenty-eight years, with exophthalmic goiter.

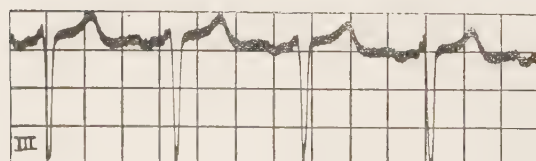
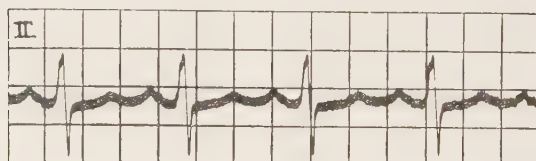
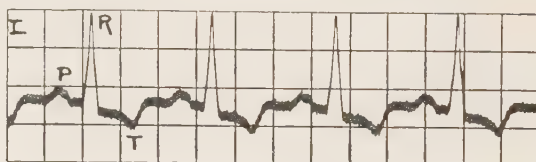


FIG. 254.—Record showing inversion of the T waves in lead I and marked preponderance of the left ventricle. The P waves in lead III are inverted. Record obtained from a man, aged forty-four years, with hypertensive heart disease and aortic sclerosis with dilatation.

and occasionally it is seen in the early period following coronary thrombosis. Similar T waves have been recorded after experimental ligation of the coronary artery in dogs (Figs. 252 and 253).

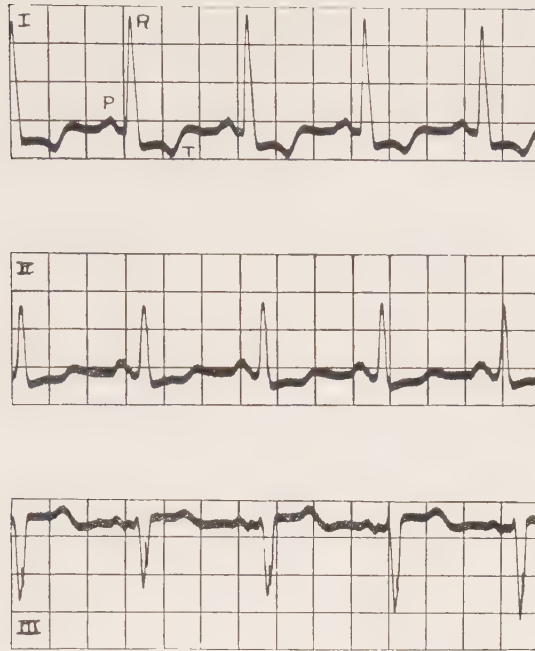


FIG. 255.—Record showing inversions of the T waves in lead I. The T waves in lead II are diphasic. Note the phasic variation in amplitude, and the notching of the QRS complexes, in lead III. Record obtained from a woman, aged sixty years, with hypertensive heart disease.

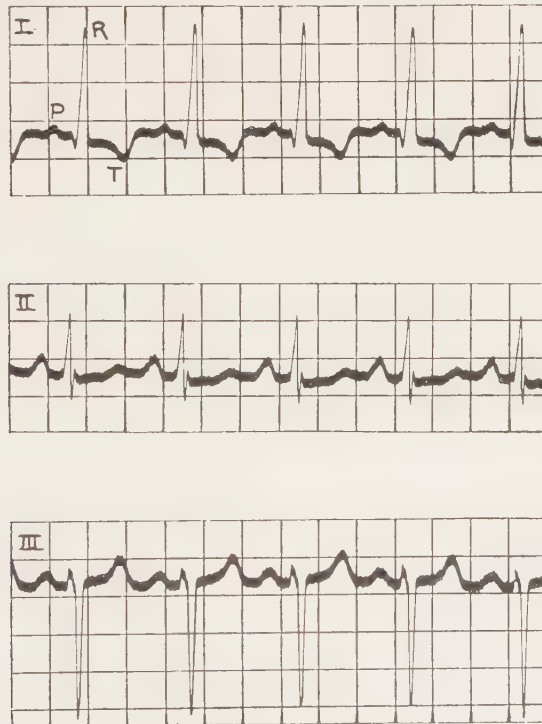


FIG. 256.—Record showing inversion of the T waves in lead I. There is marked preponderance of the left ventricle. Record obtained from a man, aged forty-three years, with hypertensive heart disease.

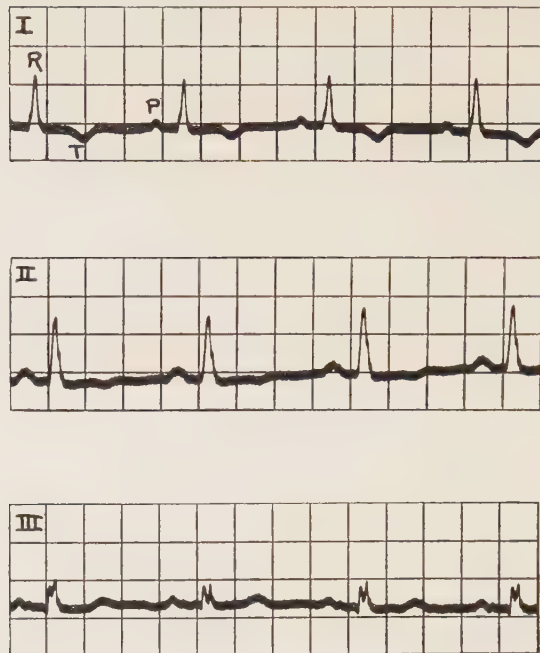


FIG. 257.—Record showing inversions of the T waves in lead I and II. Note, in lead III, the notching and low amplitude of the QRS complexes. Record obtained from a man, aged fifty-one years, with hypertensive heart disease.



FIG. 258.—Record showing inversions of the T waves in leads I and II. The T waves in lead III are diphasic. Marked notching of the QRS complexes in lead III occurs. Record obtained from a man, aged forty-eight years, with hypertensive heart disease.



FIG. 259.—Record showing inversion of the T waves in leads I and II. Note in lead III the “M” notching and the low amplitude of the QRS complexes. Record obtained from a woman, aged fifty-four years, with hypertensive heart disease.

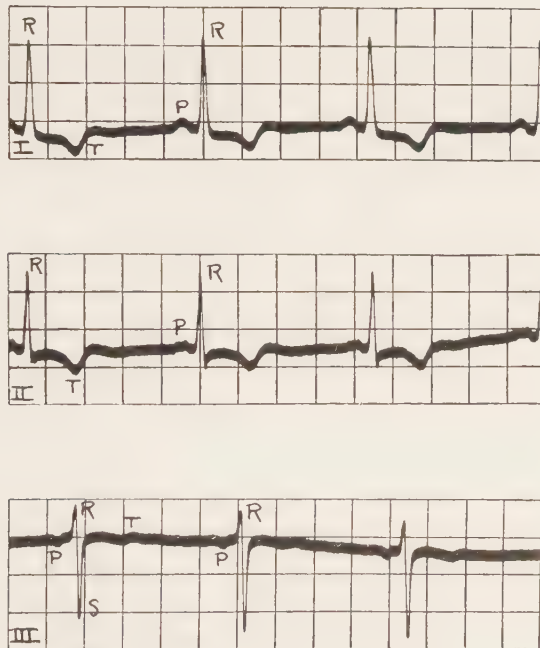


FIG. 260.—Record showing T waves which are of considerable amplitude and are inverted in leads I and II. They are of low amplitude and are diphasic in lead III. There is marked preponderance of the left ventricle, as evidenced by the deeply depressed “S” deflections in lead III. Record obtained from a man, aged sixty-three years, with coronary and aortic sclerosis accompanied by angina pectoris.

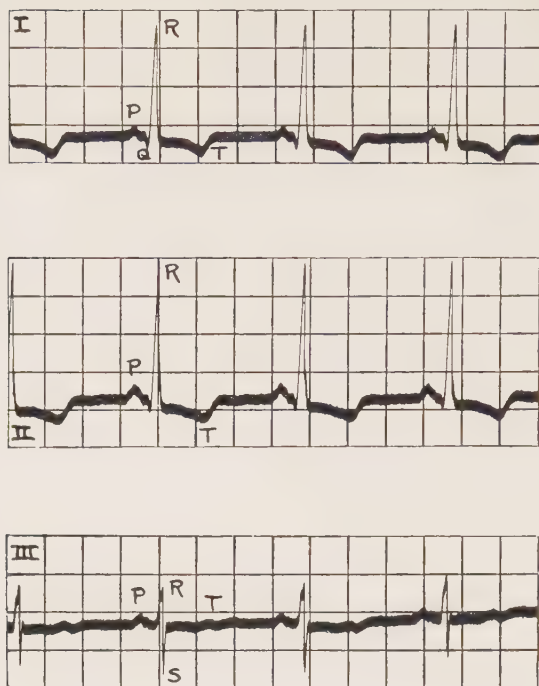


FIG. 261.—Record showing inverted T waves in leads I and II. Note the R waves of unusually high amplitude in lead II. There is moderate preponderance of the left ventricle. Record obtained from a man, aged sixty-one years, with hypertensive and coronary heart disease accompanied by angina pectoris.

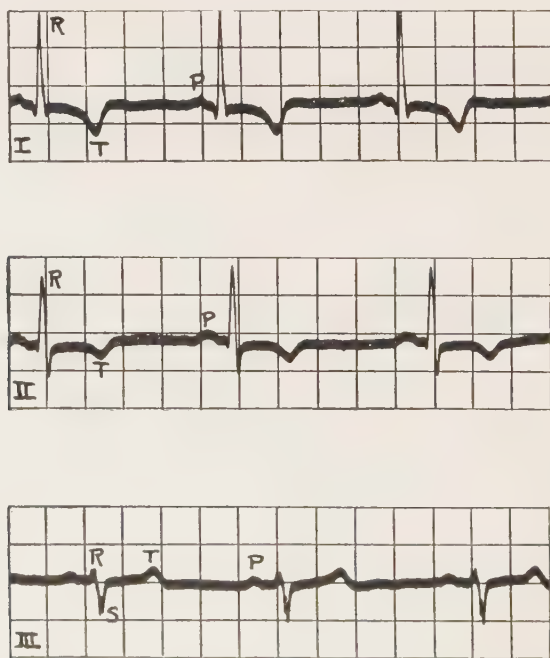


FIG. 262.—Record showing inversion of the T waves in leads I and II. Note the deep T waves in lead I. There is a moderate degree of preponderance of the left ventricle. Record obtained from a man, aged sixty-two years, with hypertensive heart disease.

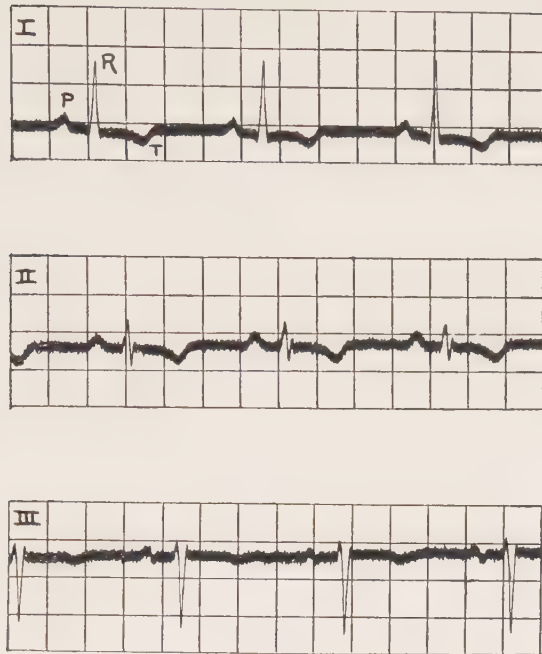


FIG. 263.—Record showing inversion of the T waves in all leads. There is preponderance of the left ventricle. Record obtained from a man, aged fifty-seven years, with hypertensive and coronary heart disease.

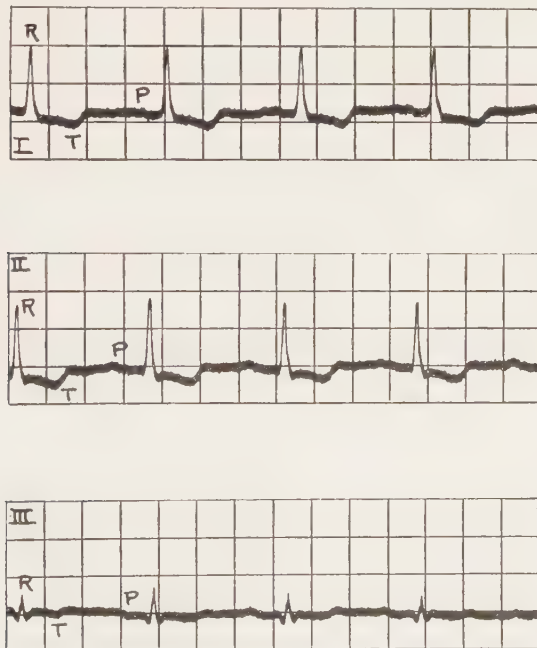


FIG. 264.—Record showing inverted T waves in all leads. Those in leads I and II have considerable amplitude. Record obtained from a woman, aged fifty-two years, with hypertensive heart disease.

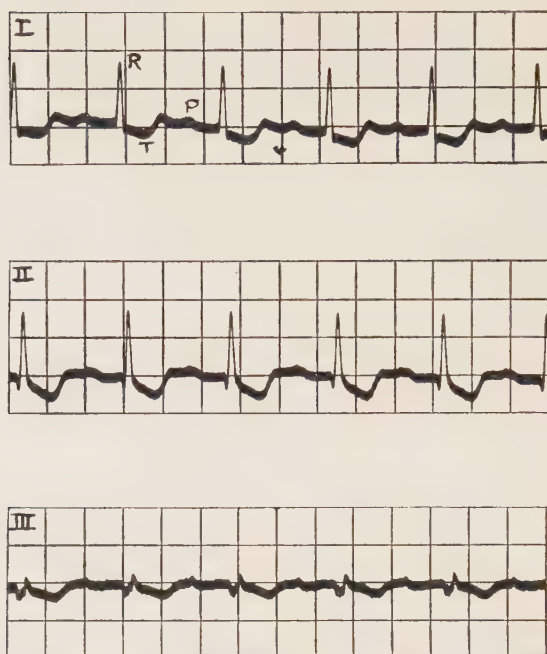


FIG. 265.—Record showing inverted T waves in all leads. Note the low amplitude of the QRS complexes in lead III. Record obtained from a woman, aged sixty-four years, with coronary sclerosis.

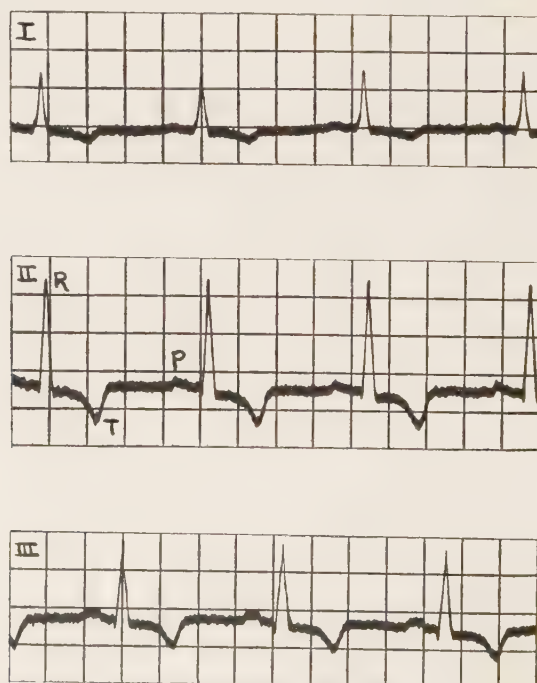


FIG. 266.—Record showing inverted T waves in all leads. The T waves in lead II, in addition to being inverted, are considerably exaggerated in amplitude. Record obtained from a man, aged thirty-nine years, who had chronic rheumatic endocarditis with aortic stenosis and insufficiency.

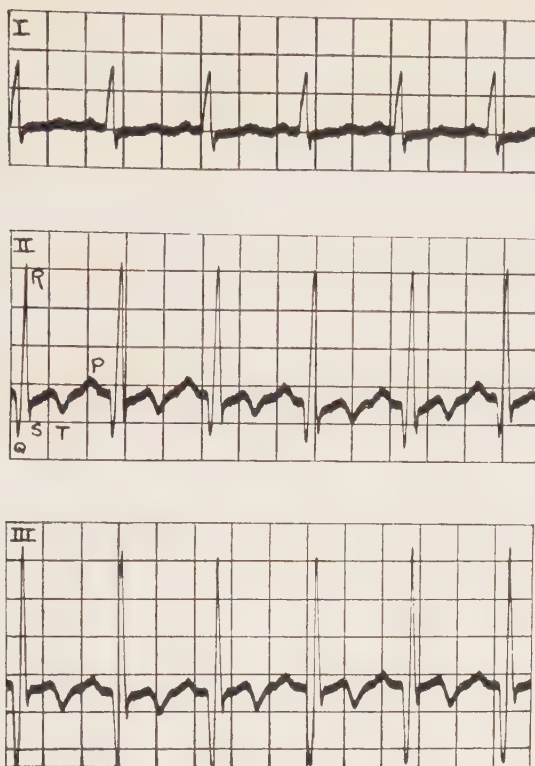


FIG. 267.—Record showing inverted T waves in leads II and III. Note the high, upright QRS complexes in leads II and III. Record obtained from a man, aged twenty-five years, who had chronic rheumatic endocarditis with aortic insufficiency.

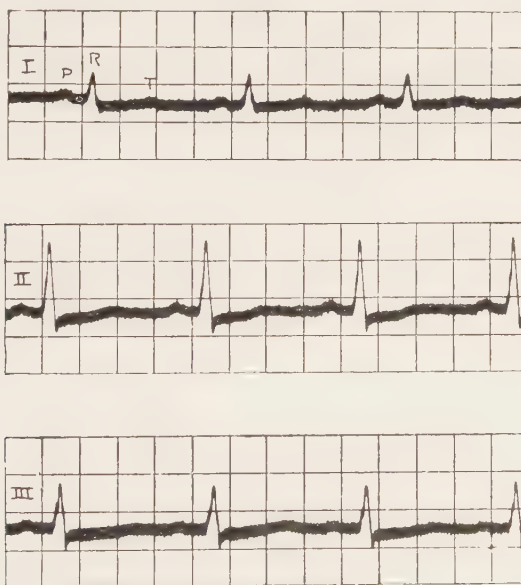


FIG. 268. —The three records presented here are those of a man, aged forty-one years, with syphilitic aortitis and aortic insufficiency associated with severe seizures of cardiac pain. The records were obtained on two consecutive days. The record taken between attacks shows the T waves to be upright in leads I and II and virtually iso-electric in lead III. There is also slurring of the QRS complexes in leads I and II and slight notching of the ascending limb in lead III.

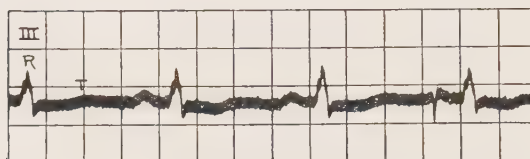
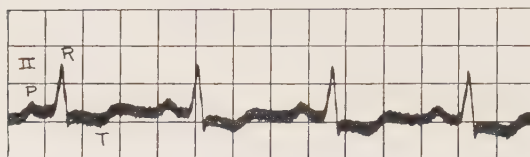
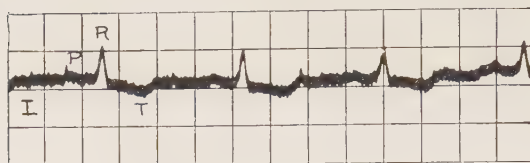


FIG. 269.—Record obtained during a severe paroxysm of cardiac pain. This shows the T waves inverted in leads I and II and diphasic in lead III. (See also Fig. 268.)

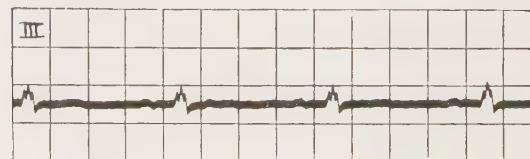
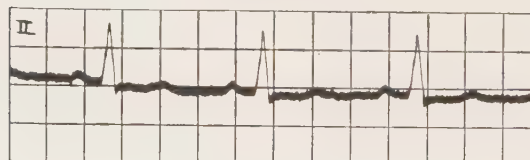
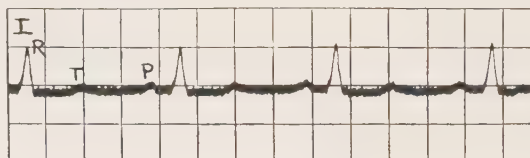


FIG. 270.—Record obtained shortly after the paroxysm of cardiac pain. This shows the T waves upright in all leads. Note the peculiar notching of the QRS complexes in lead III. (See also Figs. 268 and 269.)

Pardee has described a change in the T wave which is frequently observed in coronary thrombosis. The wave is either negative or diphasic and occurs before the completion of the R wave, deforming its contour (Figs. 271 to 274).

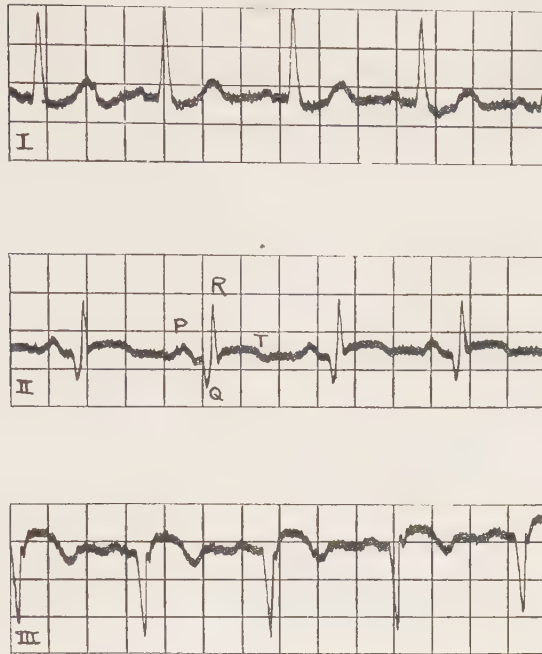


FIG. 271.—Record showing the high take-off of the inverted T waves in leads II and III (so-called coronary T waves). There is marked preponderance of the left ventricle. Record obtained from a woman, aged sixty-four years, with coronary sclerosis accompanied by angina pectoris. There was a history suggestive of recent coronary thrombosis.

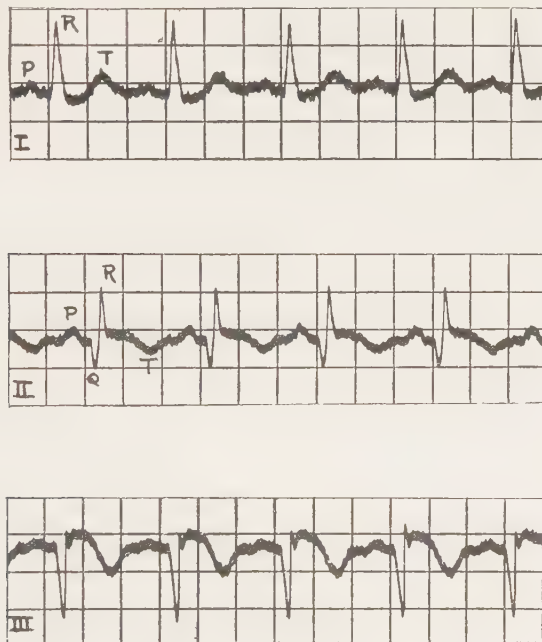


FIG. 272.—Record showing inverted T waves in leads II and III. Note the high take-off of the T waves in lead II, before completion of the QRS complexes, conforming to the characteristics of the so-called coronary T waves. Also note the deep, flaring character of the inverted T waves in lead III. There is rather marked preponderance of the left ventricle. Record obtained from a woman, aged sixty-four years, with coronary sclerosis and recent coronary thrombosis.

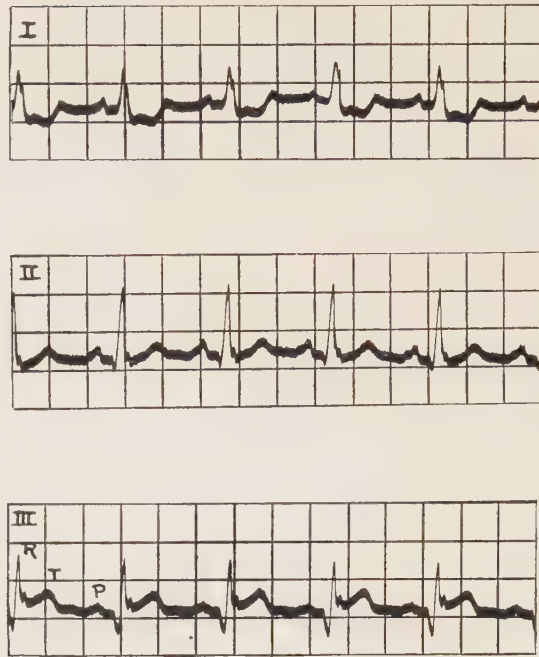


FIG. 273.—Record showing a high take-off of the T waves in lead III, similar to the so-called coronary T waves. Record obtained from a woman, aged sixty years, with coronary sclerosis accompanied by angina pectoris.

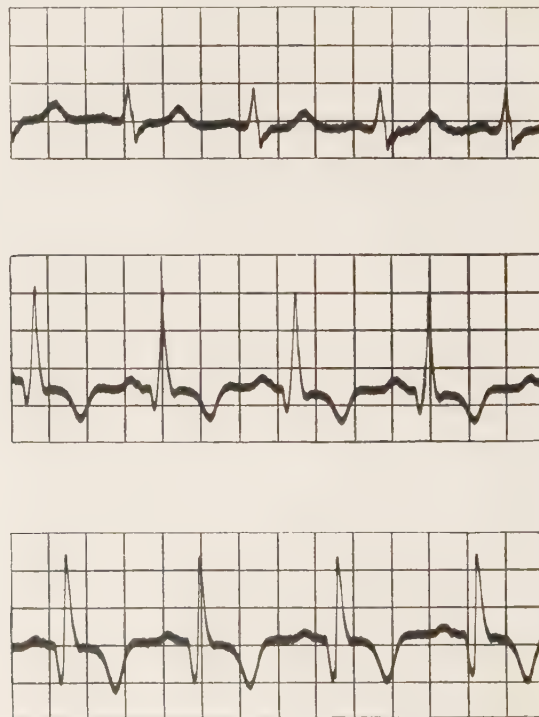


FIG. 274.—Record showing the so-called coronary T waves. Record obtained from a man, aged fifty-nine years, with coronary sclerosis.

CHANGES IN DIRECTION OF THE T WAVE

Inversion (negativity) of the T wave occurs under a variety of conditions. It may be brought on by the administration of digitalis, and at times it follows the cooling of the heart which may occur during the drinking of considerable quantities of ice water. Under these conditions, the inversion is a transient phenomenon.

However, as a constant characteristic of great clinical importance, inversion of the T wave frequently occurs independently of the afore-mentioned states (Figs. 254 to 271). It has been previously stated that the explanation of the exact nature of the normal T wave is still hypothetic except for the fact that it is known to be a ventricular event. Likewise, the permanent inversion of the wave, as seen in pathologic states, is not fully understood. Some knowledge of this abnormality, however, has been obtained through the correlation of clinical and pathologic material. In the analysis of necropsy material from a group of 130 cases, two striking observations were made: (1) coronary sclerosis of a degree sufficient to interfere materially with cardiac nutrition occurred in 40 per cent of the cases; cardiac hypertrophy great enough for the heart to weigh 400 gm. or more was present in 55 per cent of the 130 cases. It is possible that marked cardiac hypertrophy, or coronary disease, or both, may be capable of producing changes in the state of excitation of the ventricles and of influencing the refractory period.

Clinical experience with large groups of patients who have cardiac disease that is accompanied by inversion of the T wave in certain leads of the electrocardiogram has revealed a high incidence of coronary disease and of rather marked hypertrophy. This has been observed especially with hypertension, aortic stenosis, and aortic insufficiency. Other cardiopathies also display electrocardiograms with T-wave negativity, but much less frequently.

T-wave negativity may occur, with varying significance, in isolated or in combined leads of the electrocardiogram. It does not occur in lead II alone or in combined leads I and III.

Experience has shown that the occurrence of T-wave negativity according to leads is of definite clinical importance and such inversions are subsequently referred to as "significant" T-wave negativity. T-wave negativity in lead I, in leads I and II and in leads I, II and III is of the greatest importance, whereas that which occurs in leads II and III is important, but to a lesser degree. T-wave negativity in lead III alone occurs frequently in normal hearts.

Clinicians must use caution in the evaluation of T-wave negativity and must make sure that the changes are not the result of the administration of digitalis or perhaps of other temporary influences. Cases are occasionally observed in which T-wave negativity in leads II and III occurs as a transient phenomenon and it is advisable to repeat the records in these cases before placing too much emphasis on the observations.

In order to demonstrate the prognostic value of significant T-wave negativity, tables have been prepared which show the associated cardiac mortality. There are no means whereby it can be foretold whether or not a given case will exhibit T-wave negativity in the electrocardiogram. I have emphasized repeatedly that these changes are important because they are an indication of serious heart disease and because they are attended by a high and relatively early cardiac mortality.

The general significance of T-wave negativity is evident from the data contained in

Tables 13 to 26. Table 13 is illustrative of T-wave negativity in lead I, associated with coronary disease. Two hundred seventy-two patients comprised this group and 181 of

TABLE 13
MORTALITY IN CASES OF CORONARY DISEASE WITH T-WAVE NEGATIVITY IN LEAD I

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
30-39	1	1						1	100
40-49	27	22	5	15	56	15		12	44
50-59	96	80	16	62	65	18	2	32	33
60-69	109	92	17	78	72	18	1	30	27
70-79	36	29	7	23	64	11	4	9	25
80-89	3	2	1	3	100	2			
Total and average.	272	226	46	181	67	17	7	84	31

them (67 per cent) died of heart disease within an average of seventeen months following examination.

The statistical data regarding T-wave negativity in leads I and II, associated with coronary disease, are presented in Table 14. There were 116 patients, of whom seventy-five

TABLE 14
MORTALITY IN CASES OF CORONARY DISEASE WITH T-WAVE NEGATIVITY IN LEADS I AND II

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
30-39	1	1		1	100	3			
40-49	15	13	2	8	53	32		7	47
50-59	47	38	9	30	64	17		17	36
60-69	49	39	10	33	67	11	3	13	27
70-79	4	3	1	3	75	3		1	25
Total and average.	116	94	22	75	65	12	3	38	33

(65 per cent) died of heart disease within an average of twelve months following examination. T-wave negativity in all leads, associated with coronary disease, is shown in Table 15. There were seventy-six patients, and fifty of them (66 per cent) died of heart disease within an average of twelve months following examination.

The mortality accompanying T-wave negativity in leads II and III is shown in Table 16. In a group of 119 patients, fifty-nine (50 per cent) died of heart disease within an average of twenty-four months following examination.

TABLE 15
MORTALITY IN CASES OF CORONARY DISEASE WITH T-WAVE NEGATIVITY IN LEADS I, II, AND III

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
40-49	5	5		2	40	18		3	60
50-59	30	28	2	19	63	9		11	37
60-69	33	26	7	23	70	15	2	8	27
70-79	8	6	2	6	75	10	1	1	13
Total and average.	76	65	11	50	66	12	3	23	30

Data fairly analogous to those given in Table 16 are obtained in the investigation of T-wave negativity that is associated with hypertensive heart disease. Table 17 shows the mortality that accompanies T-wave negativity in lead I, in the presence of hypertensive heart disease. In a group of 463 patients, 260 (56 per cent) died of heart disease within an

TABLE 16
MORTALITY IN CASES OF CORONARY DISEASE WITH T-WAVE NEGATIVITY IN LEADS II AND III

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
30-39	1	1					1		
40-49	17	14	3	12	71	21		5	29
50-59	38	30	8	17	45	35		21	55
60-69	47	42	5	21	45	18	3	23	49
70-79	14	11	3	8	57	11		6	43
80-89	2	1	1	1	50	23		1	50
Total and average.	119	99	20	59	50	24	4	56	47

average of nineteen months following examination. T-wave negativity in leads I and II, associated with hypertensive heart disease, is shown in Table 18. In a group of 281 patients, 166 (59 per cent) died of heart disease within an average of thirteen months following examination. The data concerning T-wave negativity in all leads when accompanied by hyper-

tensive heart disease is found in Table 19. There were 135 patients, and ninety-one (67 per cent) died of heart disease within an average of twelve months following examination.

TABLE 17
MORTALITY IN CASES OF HYPERTENSIVE HEART DISEASE WITH T-WAVE NEGATIVITY IN LEAD I

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
20-29	4	3	1	2	50	6	2		
30-39	14	12	2	7	50	20	4	3	21
40-49	100	63	37	59	59	18	26	15	15
50-59	182	123	59	100	55	18	28	54	30
60-69	136	93	43	76	56	18	22	38	28
70-79	24	17	7	13	54	36	6	5	28
80-89	3	3		3	100	23			
Total and average.	463	314	149	260	56	19	88	115	25

Table 20 shows the mortality data concerning T-wave negativity in leads II and III associated with hypertensive heart disease. This group comprised 140 patients, of whom sixty-

TABLE 18
MORTALITY IN CASES OF HYPERTENSIVE HEART DISEASE WITH T-WAVE NEGATIVITY IN LEADS I AND II

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
20-29	2	1	1	1	50	8	1		
30-39	25	17	8	16	64	7	6	3	12
40-49	59	38	21	36	61	12	14	9	15
50-59	110	80	30	57	52	15	28	25	23
60-69	77	51	26	52	68	15	7	18	23
70-79	6	5	1	3	50	4	2	1	17
80-89	2	1	1	1	50	13		1	50
Total and average.	281	193	88	166	59	13	58	57	20

one (44 per cent) died of heart disease within an average of seventeen months following examination.

TABLE 19

MORTALITY IN CASES OF HYPERTENSIVE HEART DISEASE WITH T-WAVE NEGATIVITY IN LEADS I, II, AND III

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
20-29	6	3	3	3	50	15	1	2	33
30-39	11	6	5	6	55	9	3	2	18
40-49	33	18	15	21	64	7	6	6	18
50-59	46	38	8	32	70	12	6	8	17
60-69	37	24	13	27	73	15	2	8	22
70-79	2	2		2	100	9			
Total and average.	135	91	44	91	67	12	18	26	19

TABLE 20

MORTALITY IN CASES OF HYPERTENSIVE HEART DISEASE WITH T-WAVE NEGATIVITY IN LEADS II AND III

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
20-29	6	2	4	2	33	6	1	3	50
30-39	12	7	5	6	50	4	3	3	25
40-49	31	22	9	16	52	20	2	13	42
50-59	48	29	19	19	40	16	4	25	52
60-69	36	23	13	15	42	20	6	15	41
70-79	6	5	1	2	34	15		4	66
80-89	1	1		1	100	20			
Total and average.	140	89	51	61	44	17	16	63	45

The constantly high mortality averages in these cases are striking. They are not the result of one study but of repeated studies that were made over a period of fourteen years.

To emphasize further the importance of T-wave negativity, I will present other tables. In one of these tables, for instance, will be shown the mortality in a group of patients with a certain cardiac disease; in another table will appear the mortality among the patients of the same group who exhibited the graphic changes under discussion. In this manner, comparison will be possible.

In Table 21 are shown the statistics of mortality of a group of 347 patients with mitral stenosis; the figures were compiled regardless of electrocardiographic changes. One hundred twenty-eight of these patients (37 per cent) died of heart disease within an average of

sixteen months following examination. Table 22 represents the portion of the group included in Table 21 that exhibited significant T-wave negativity. There were forty-three patients, and twenty-seven (63 per cent) died of heart disease within an average of twelve

TABLE 21
MORTALITY IN CASES OF MITRAL STENOSIS

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
0-9	6	2	4	2	33	5	1	3	50
10-19	24	9	15	9	38	20		15	62
20-29	83	25	58	33	40	20	7	43	52
30-39	115	44	71	34	30	24	12	69	60
40-49	86	20	66	32	37	14	4	50	58
50-59	28	10	18	16	57	17	1	11	39
60-69	5	2	3	2	40	11		3	60
Total and average.	347	112	235	128	37	16	25	194	56

months following examination. The contrast in the figures of mortality in the two groups is striking and the constancy of this discrepancy in all types of cardiopathy studied is especially interesting.

TABLE 22
MORTALITY IN CASES OF MITRAL STENOSIS WITH SIGNIFICANT T-WAVE NEGATIVITY

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
10-19	1		1					1	100
20-29	15	7	8	9	60	8	1	5	33
30-39	10	4	6	5	50	13	2	3	30
40-49	12	5	7	9	75	11		3	25
50-59	4	2	2	3	75	17		1	25
60-69	1	1		1	100	13			
Total and average.	43	19	24	27	63	12	3	13	30

In Table 23 are shown the data concerning 200 cases of rheumatic aortic insufficiency, grouped regardless of graphic changes. There were seventy-eight (39 per cent) cardiac deaths within an average of sixteen months following examination. This group is compared,

TABLE 23
MORTALITY IN CASES OF RHEUMATIC AORTIC INSUFFICIENCY

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
10-19	23	17	6	8	35	9	1	14	61
20-29	55	50	5	22	40	21	6	27	49
30-39	52	44	8	13	25	14	10	29	56
40-49	29	20	9	17	59	20	2	10	34
50-59	28	21	7	11	39	14	5	12	43
60-69	10	8	2	5	50	3	2	3	30
70-79	3	3		2	67	5	1		
Total and average.	200	163	37	78	39	16	27	95	48

in Table 24, with those cases in the same group in which significant T-wave negativity was displayed in the graphic records. There were sixty-two patients, and thirty-five (56 per cent) died of heart disease within an average of fifteen months following examination.

TABLE 24
MORTALITY IN CASES OF RHEUMATIC AORTIC INSUFFICIENCY WITH SIGNIFICANT T-WAVE NEGATIVITY

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
10-19	2	2						2	100
20-29	22	21	1	13	59	18	1	8	36
30-39	16	13	3	5	31	10	2	9	56
40-49	9	8	1	9	100	24			
50-59	9	9		4	44	7		5	56
60-69	3	3		3	100	3			
70-79	1	1		1	100	3			
Total and average.	62	57	5	35	56	15	3	24	39

In the total group of 137 cases of syphilitic aortic insufficiency, reviewed regardless of electrocardiographic changes (Table 25) death occurred from heart disease in sixty-three (46 per cent) within an average of sixteen months following examination. In selecting from this group those cases in which significant T-wave negativity was shown, striking contrasts in the averages of mortality again are evident. These data are shown in Table

TABLE 25
MORTALITY IN CASES OF SYPHILITIC AORTIC INSUFFICIENCY

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
20-29	8	7	1	2	25	1	2	4	50
30-39	30	26	4	11	37	12		19	63
40-49	58	52	6	30	52	17	4	24	41
50-59	29	27	2	12	41	18	1	16	55
60-69	12	10	2	8	67	13	1	3	25
Total and average.	137	122	15	63	46	16	8	66	48

26. There were forty-two patients, and thirty-two (76 per cent) died of heart disease within an average of eleven months following examination. The data presented warrant attaching importance to significant T-wave negativity in the electrocardiogram.

TABLE 26
MORTALITY IN CASES OF SYPHILITIC AORTIC INSUFFICIENCY WITH SIGNIFICANT T-WAVE NEGATIVITY

Decade.	Total cases.	Sex.		Cardiac deaths.			Deaths from other causes.	Patients living.	
		Males.	Females.	Cases.	Per cent.	Average time after examination, months.		Number.	Per cent.
20-29	1	1		1	100	2			
30-39	6	5	1	5	83	9		1	17
40-49	24	22	2	18	75	13		6	25
50-59	7	7		4	57	8	1	2	29
60-69	4	4		4	100	12			
Total and average.	42	39	3	32	76	11	1	9	21

Changes which occur with coronary thrombosis will be discussed in a later chapter.

DIPHASIC T WAVES

Knowledge regarding diphasic T waves is still limited. Their occurrence as constant phenomena is probably significant, for they have been observed as precursors of permanent negativity.

LOW-VOLTAGE WAVES

Considerable prognostic importance has been accorded electrocardiograms which display waves of low voltage or low amplitude. Such records are shown in Figures 250 and 251. Whether this prognostic importance is justified is doubtful, for Willius and

Killins, in a study of cases in which this abnormality was recorded, did not find appreciable increase in cardiac mortality when other significant graphic abnormalities had been excluded. The occurrence of records of low voltage in cases of heart disease in which previous graphs have been normal may be of considerable significance; however, few data regarding this transition are available at this time.

PROLONGED S-T INTERVAL

Not infrequently electrocardiograms are observed in which the S-T interval is prolonged beyond the accepted limits of normal, 0.24 to 0.28 second (Fig. 275). The exact significance of the observation is uncertain; it is observed in graphs made from apparently normal hearts as well as from hearts which display definite evidence of disease.

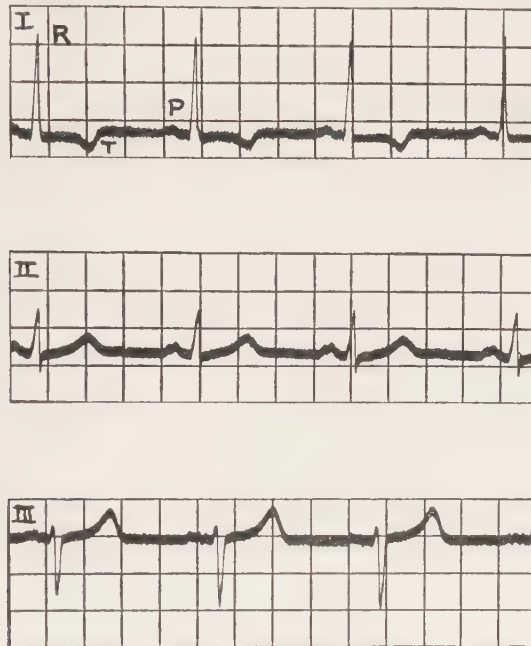


FIG. 275.—Record showing prolongation of the S-T interval to 0.32 second. The normal range varies from 0.24 to 0.28 second. The T waves in lead I are inverted and there is preponderance of the left ventricle. Record obtained from a man, aged forty-seven years, with hypertensive heart disease.

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Chapter XIV

CONGENITAL DEXTROCARDIA WITH COMPLETE SITUS TRANSVERSUS

The electrocardiograms obtained from patients who have congenital dextrocardia with complete situs transversus are characteristic. All of the waves in lead I are directed downward, whereas their normal direction is the opposite. The waves are normal in contour and bear their proper relationship one to the other. The records of such cases are

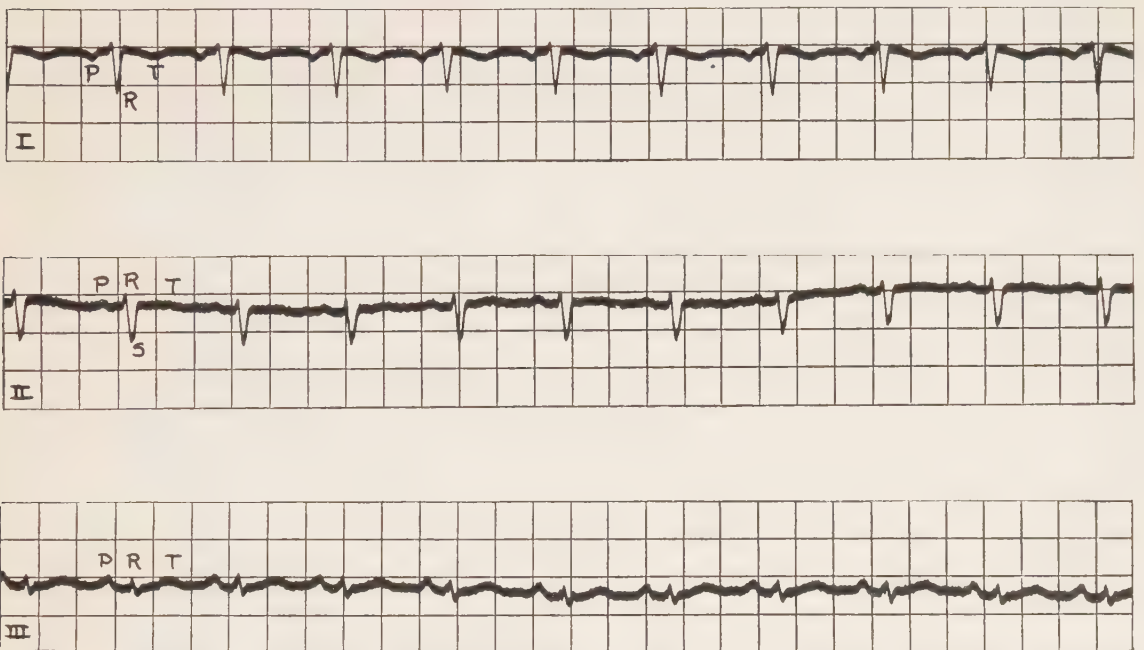


FIG. 276.—Record, from a patient with congenital dextrocardia and complete situs transversus, showing complete inversion of all the complexes in lead I. The patient was a man aged fifty-four years.

shown in Figures 276 to 279. These graphic characteristics are of distinct value in the differentiation of congenital against acquired displacement of the heart.

In this connection, it is of interest to refer to Figure 135, the record obtained from an infant six days old in whom necropsy revealed complete transposition of all the abdominal and thoracic viscera except the ventricles of the heart. The ventricular complexes in lead I are not inverted, because the ventricles maintained their proper relationships to each other.

When acquired organic cardiac disease is associated with this congenital anomaly any of the previously described electrocardiographic abnormalities may be displayed.

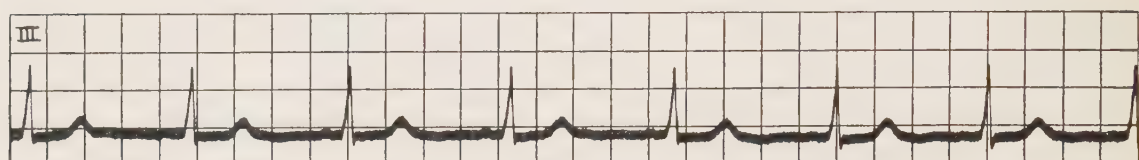
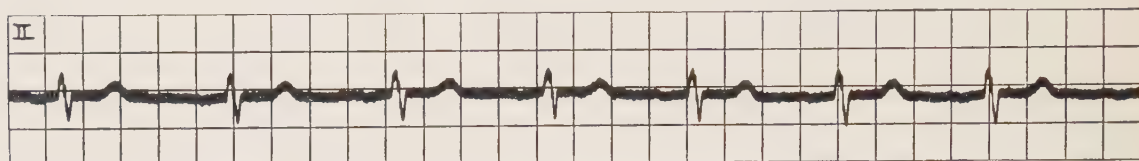
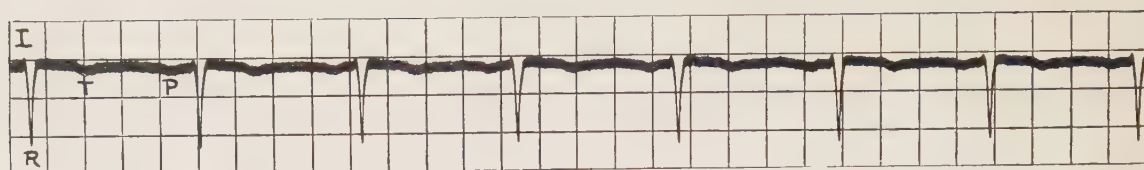


FIG. 277.—Record of a woman, aged thirty-eight years, with congenital dextrocardia and complete situs transversus. Note the inversion of all waves in lead I.

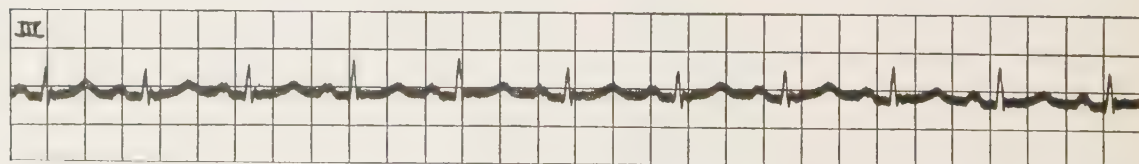
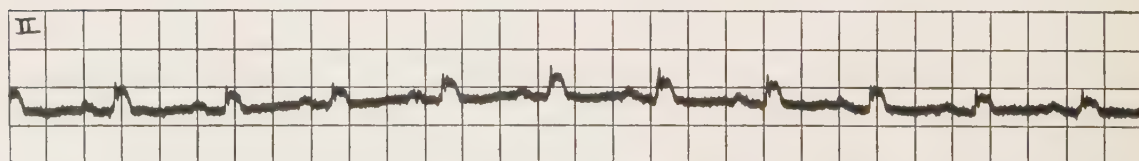
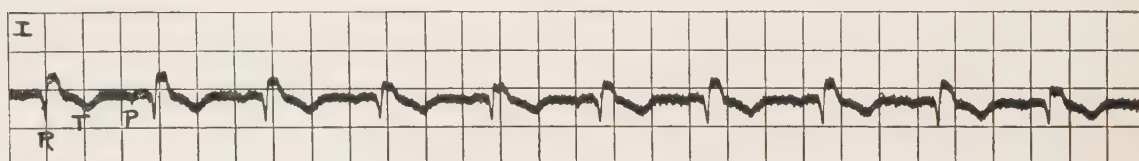


FIG. 278.—Record of a man, aged forty-three years, who had congenital dextrocardia with complete situs transversus. There is inversion of all the complexes in lead I. Note the peculiar notching of the QRS complexes in leads I and II.

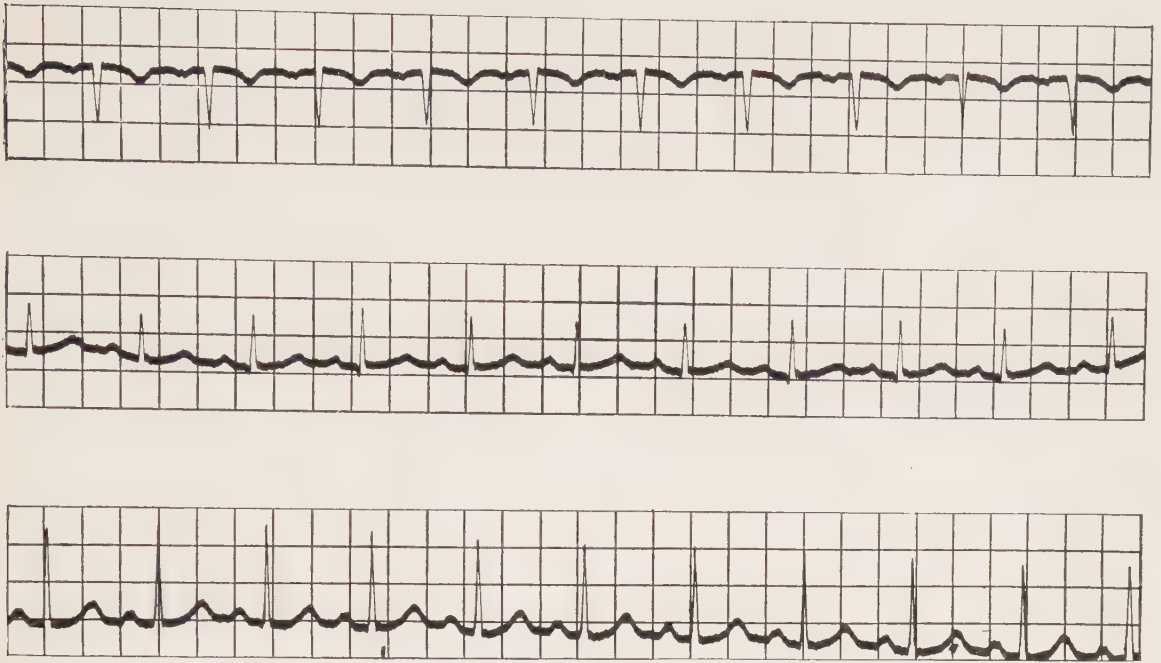


FIG. 279.—Record of a man, aged twenty-seven years, with congenital dextrocardia and complete situs transversus. The complexes in lead I are all inverted.

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Chapter XV

THROMBOSIS OF THE CORONARY ARTERIES OR THEIR BRANCHES

Some of the most spectacular graphic changes have been observed in repeated electrocardiographic examinations of patients with coronary thrombosis. In order to detect the many transitional events that occur, frequent records are necessary. At times the electrocardiogram becomes altered within thirty minutes after occlusion of a branch of a coronary artery and again changes may not be revealed until thirty-six to forty-eight hours later.

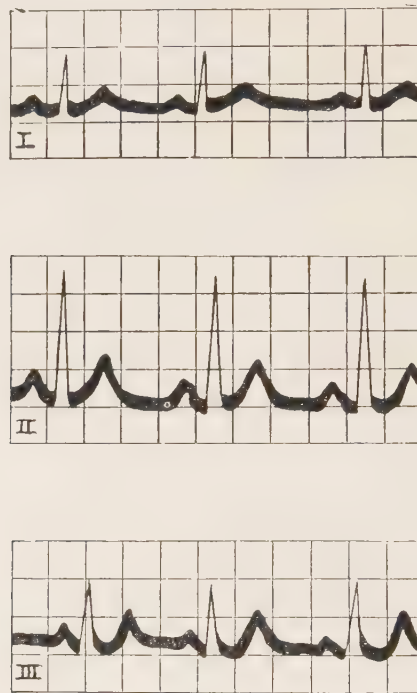


FIG. 280.—This record and the succeeding seven figures were obtained from a man, aged forty-seven years, with coronary thrombosis. The first record was obtained less than an hour after the onset of the attack. There are no striking abnormalities, but the T waves in lead II are exaggerated in amplitude.**

The most constant abnormalities observed are inversion of the T wave and the T wave with high take-off described by Pardee. The occurrence of incomplete and complete bundle-branch block is much less frequent. In rare instances the area of infarction involves the interventricular septum and complete heart-block occurs. In one case that has come under my observation, auricular fibrillation occurred within twenty minutes after the occlusion had taken place.

If patients are recovering from coronary thrombosis, it is not unusual for the electrocardiogram to return to normal within a period of six months to a year.

The records shown in this chapter are worthy of careful study. They represent five

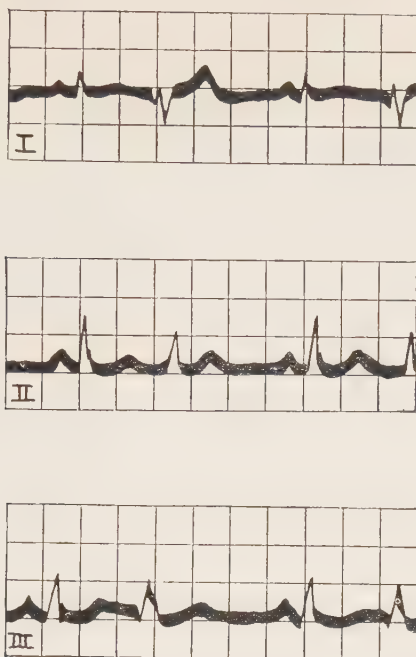


FIG. 281.—Record obtained on the following day (Fig. 280) showing only alternating premature contractions.**

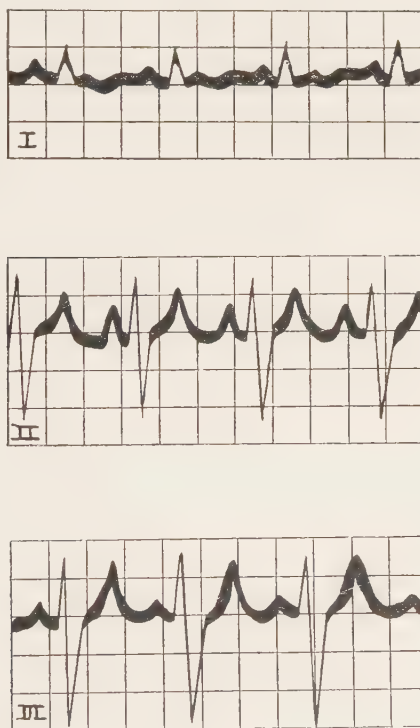


FIG. 282.—Record obtained on the following day (Fig. 281) differs greatly in all respects from the previous record. The findings conform to those of complete bundle-branch block. Note the inversion of the T components in lead I.**

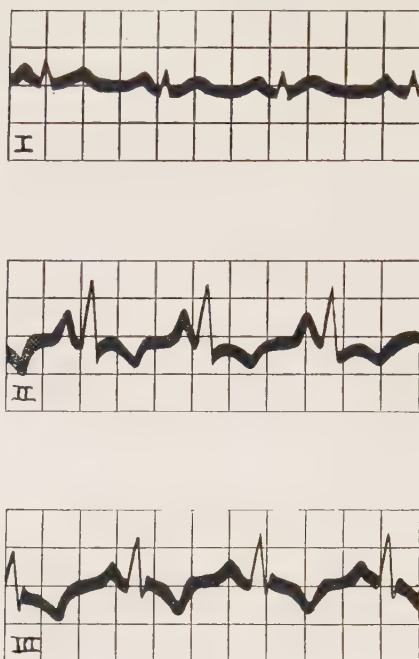


FIG. 283.—Record obtained on the following day (Fig. 282) again shows a complete change in the complexes. The bundle-branch changes have disappeared. The T waves in leads II and III are inverted, whereas those in lead I are now upright.**

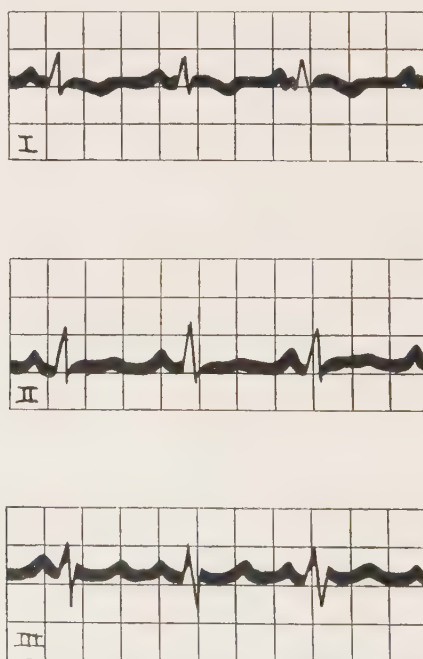


FIG. 284.—Record obtained three days later (Fig. 283) shows further changes. The T waves in lead I are inverted whereas those in leads II and III are upright.**

patients who were followed carefully, by repeated electrocardiographic examinations, and the records show extremely interesting changes.

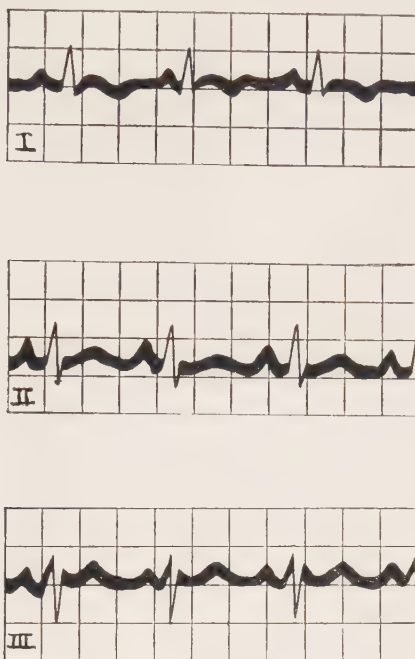


FIG. 285.—Record obtained three days later (Fig. 284) is essentially the same as the preceding record and shows well marked inversion of the T waves in lead I.**

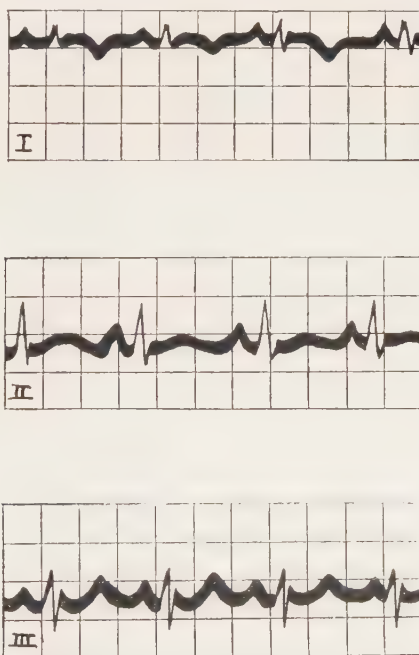


FIG. 286.—Record obtained six days later (Fig. 285) shows, in lead I, more pronounced inversion of the T waves than is apparent in the preceding record.

In the first case (Figs. 280 to 287) records were obtained less than one hour after the acute coronary closure, and other records were made almost every day for the following

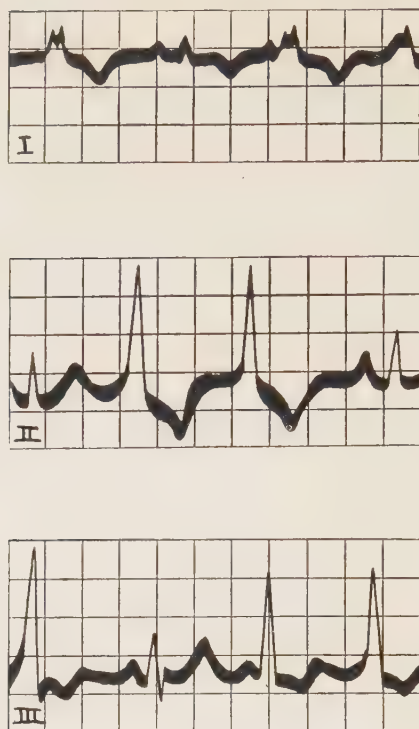


FIG. 287.—Record obtained sixteen days later (Fig. 286) shows the T waves still inverted in lead I and the presence of numerous premature ventricular contractions. The patient returned to his home. However, he gradually developed increasing cardiac insufficiency and died eight months later with congestive failure and auricular fibrillation.**

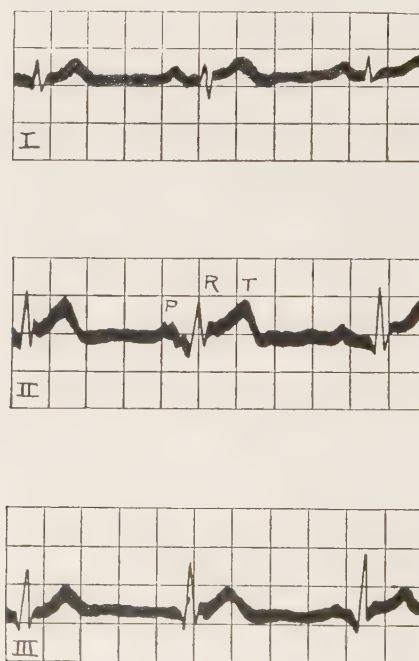


FIG. 288.—This record and the succeeding eight figures were obtained from a man, aged forty-eight years, who was observed shortly after coronary thrombosis and intermittently for three and one-half years thereafter. The first record was obtained twenty-four hours after the onset of the attack and while pain was still present. Note the T waves of high amplitude in lead II.**

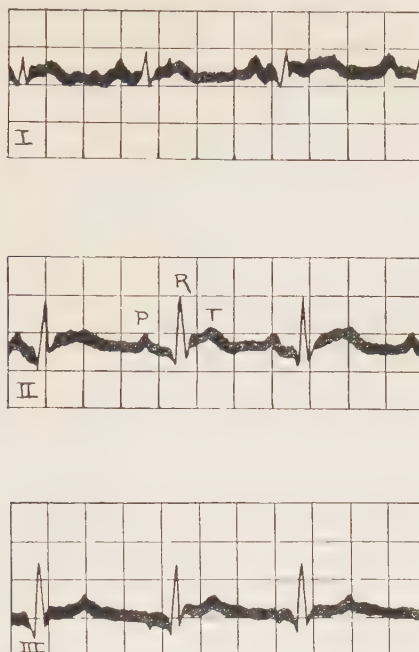


FIG. 289.—Record obtained on the following day (Fig. 288) showing the rather diphasic character of the T waves. Pain was still present at the time this record was obtained.

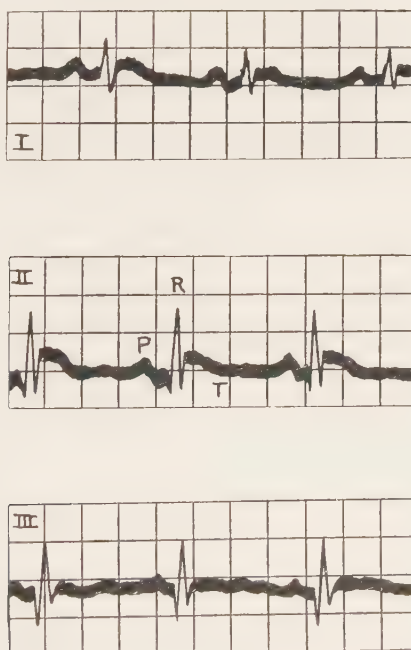


FIG. 290.—Record obtained on the following day (Fig. 289) showing lack of character of the T waves in all leads. The peculiar high take-off of the T waves in lead II is to be noted.**

month. Owing to the numerous records obtained it is possible to present only the important transitional changes that occurred. For the observation of the changes in the order

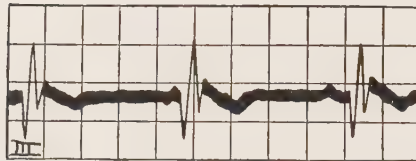
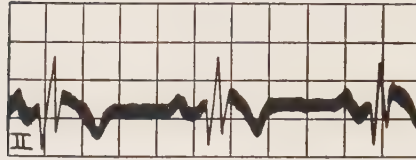
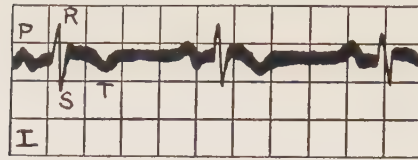


FIG. 291.—Record obtained four days later (Fig. 290) showing well marked inversions of the T waves in all leads.**

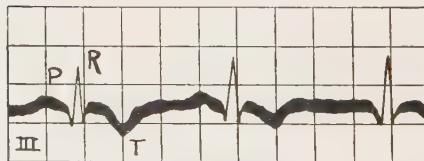
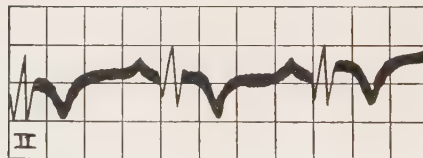
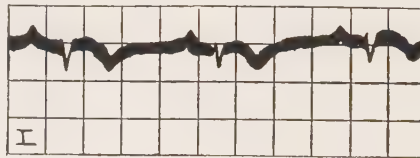


FIG. 292.—Record obtained nine days later (Fig. 291). It shows the inversions of the T waves well marked in all leads
The intervening records showed the same characteristics.**

in which they occurred, I refer the reader to the legends which accompany the electrocardiographic reproductions.

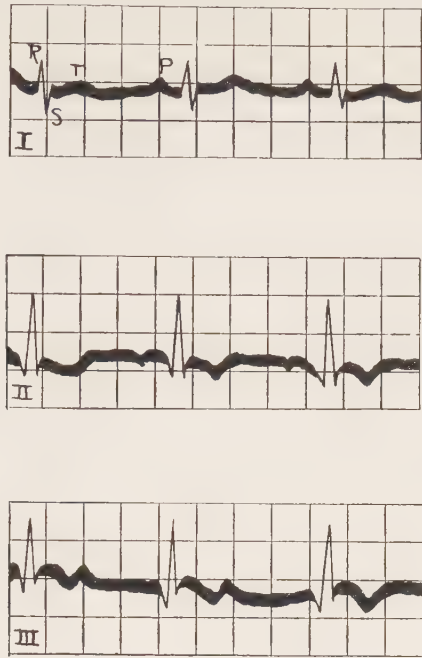


FIG. 293.—Record obtained five months later (Fig. 292). It shows inversions of the T waves in leads II and III only.**



FIG. 294.—Record obtained seven months later (Fig. 293) and a year after the acute coronary occlusion. This record is normal, showing inversions of the T waves in lead III only.

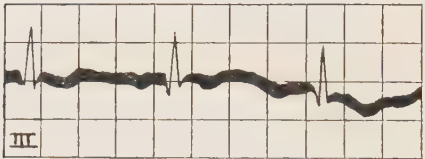
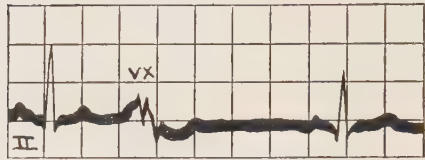
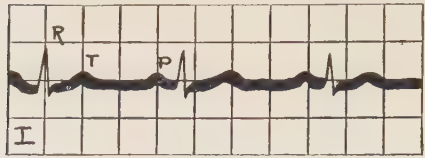


FIG. 295.—Record taken five months later (Fig. 294) showing normal tracing. One premature ventricular contraction occurs in lead II.**

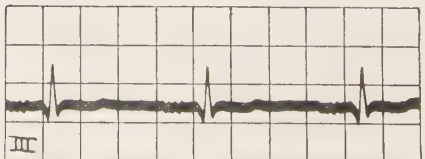
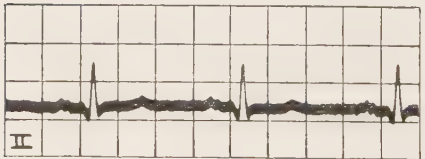
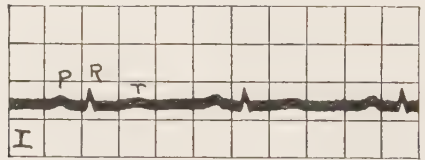


FIG. 296.—Record obtained twenty-three months later (Fig. 295). It shows a normal tracing.

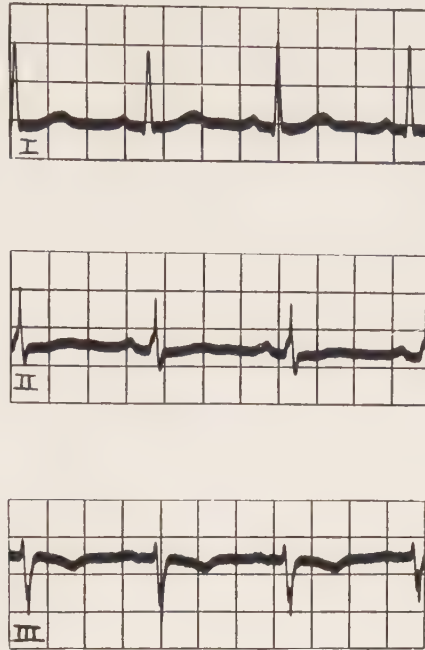


FIG. 297.—This record and those in the succeeding twelve figures were obtained from a man, aged fifty-eight years, with coronary sclerosis accompanied by angina pectoris. This record is virtually normal, showing only preponderance of the left ventricle and inversions of the T waves in lead III.

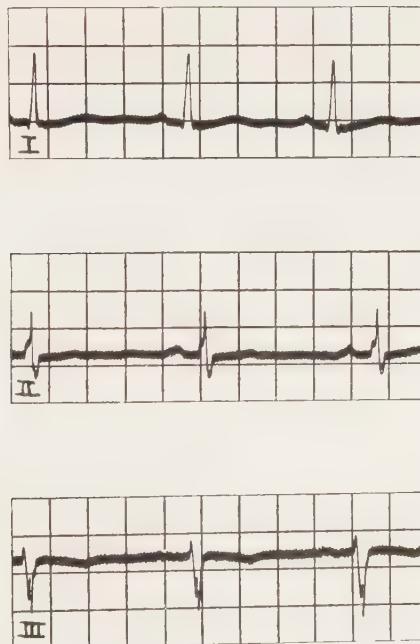


FIG. 298.—Record obtained two and one-half months later (Fig. 297). During this time the anginal attacks became more frequent. The record is virtually identical with the previous one.

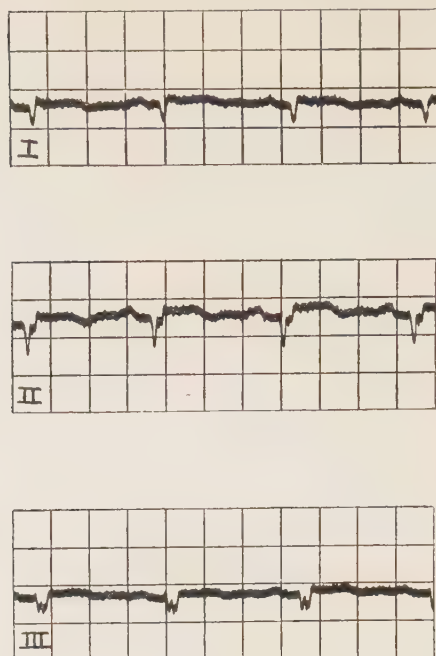


FIG. 299.—Record obtained two days later (Fig. 298) and twenty hours after the onset of a severe attack of cardiac pain which lasted fourteen hours and which was the manifestation of coronary thrombosis. Note the complete change in character of the tracing, as evidenced by QRS complexes of very low amplitude with distinct notching in leads II and III. The T waves in all leads are diphasic and are of indistinct character.

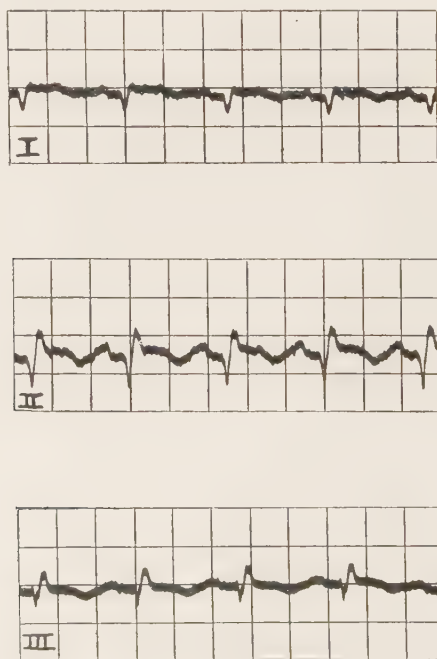


FIG. 300.—Record obtained the next day (Fig. 299), showing T waves inverted in leads II and III and diphasic in lead I.

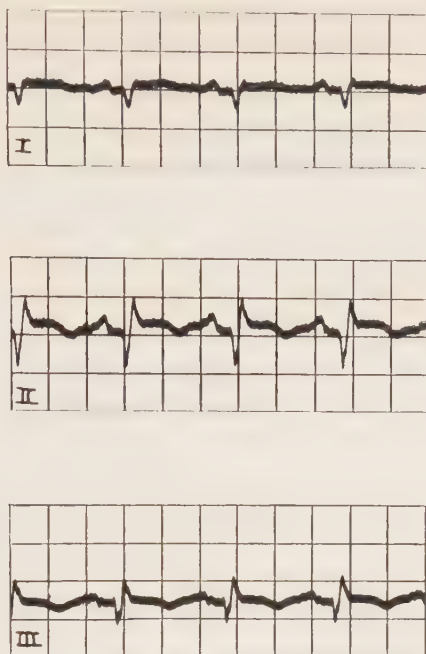


FIG. 301.—Record obtained the next day (Fig. 300). It is practically identical with the record shown in the previous figure.

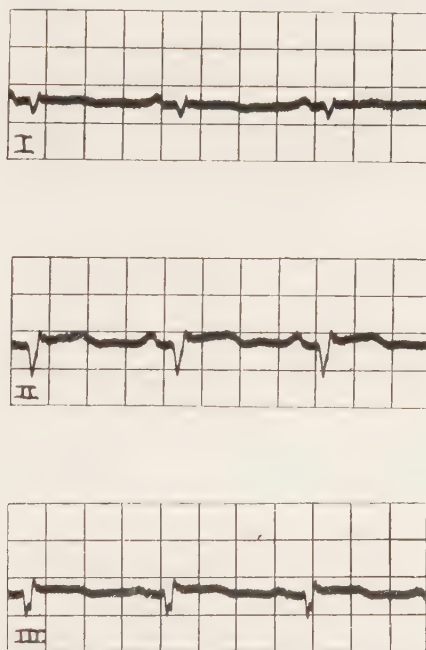


FIG. 302.—Record obtained four days later (Fig. 301). This record again shows definitely diphasic T waves in all leads

In the second case records were obtained soon after the occurrence of coronary thrombosis and at intervals for three and a half years thereafter (Figs. 288 to 296). It is interesting to note the return of a normal electrocardiographic record.

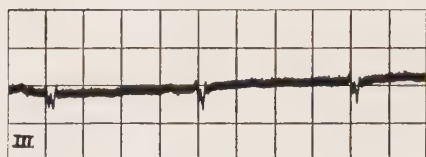
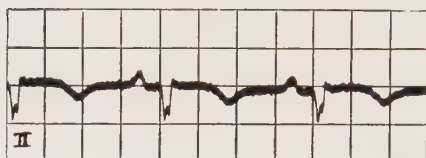
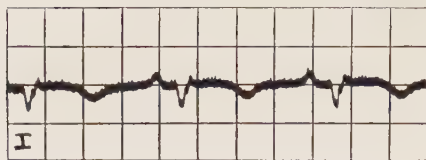


FIG. 303.—Record obtained six days later (Fig. 302). It shows the T waves inverted in leads I and II, with a prolongation of the S-T interval to 0.47 second.

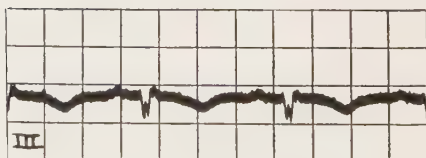
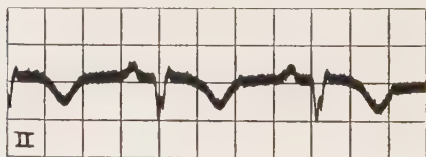
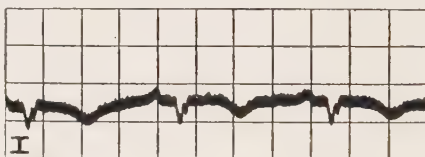


FIG. 304.—Record obtained eight days later (Fig. 303). It shows marked inversion of the T waves in all leads. The S-T interval is still prolonged.

The third case is of interest because records were obtained several months before coronary thrombosis occurred and other records were made subsequent to its occurrence (Figs. 297 to 306).

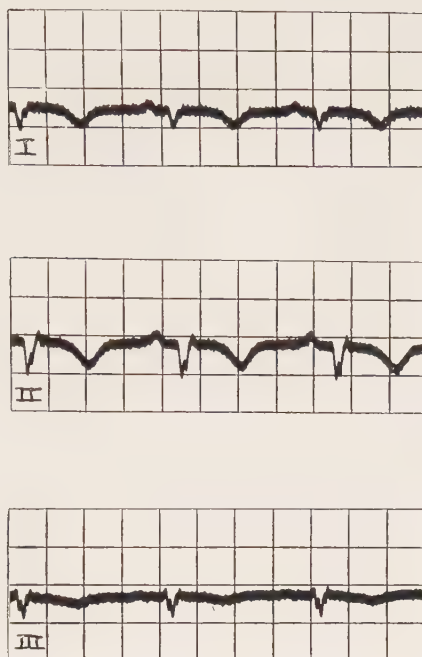


FIG. 305.—Record obtained six days later (Fig. 304). It shows the T waves still inverted in all leads. The S-T interval is less prolonged now—0.36 second.

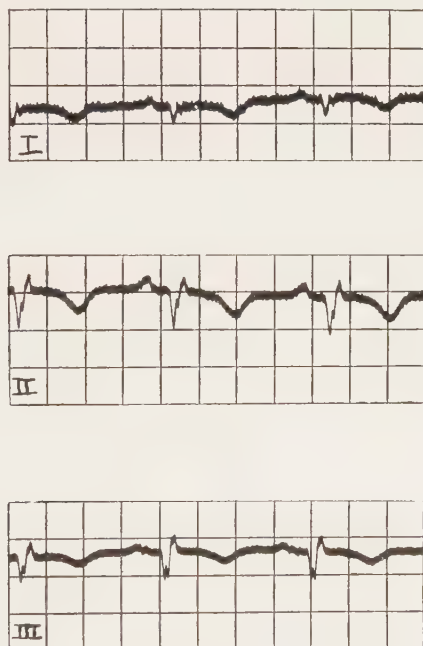


FIG. 306.—This record was obtained eight days later (Fig. 305) and shows but little change.

In the fourth case records were obtained before coronary occlusion had occurred but in the electrocardiogram changes had appeared and probably they were the result of the

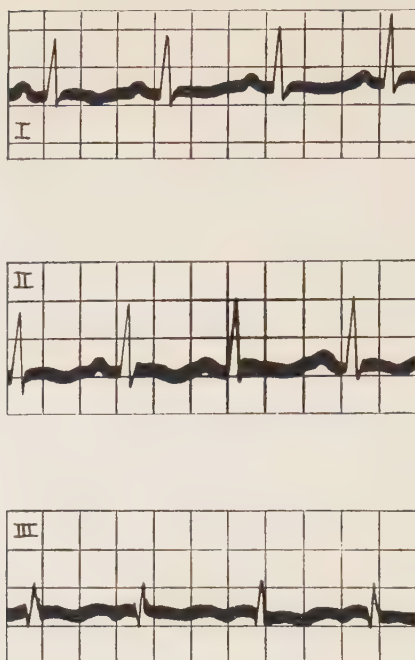


FIG. 307.—The records in this figure and in the three succeeding figures were obtained from a man, aged fifty-nine years, with coronary sclerosis accompanied by angina pectoris. This record was taken the day of the patient's admission to the hospital. Note the biphasic character of the T waves in leads I and II. The T and P waves in lead III are inverted.**

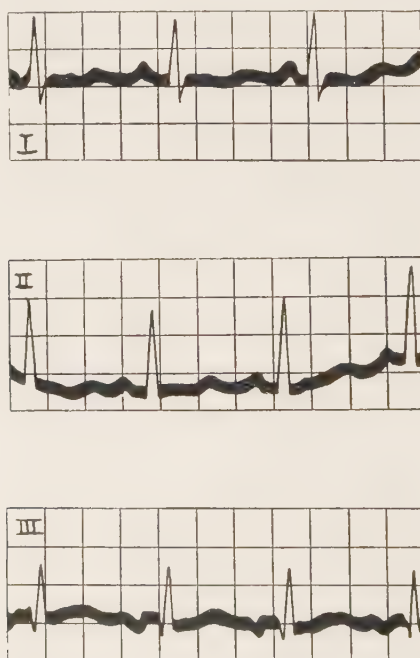


FIG. 308.—This record was obtained the following day (Fig. 307) and six hours after the onset of a severe, protracted attack of cardiac pain. This attack, with other developments, leads to the diagnosis of coronary thrombosis. Note the biphasic character of the T waves in lead I. The T waves in lead III are now upright.**

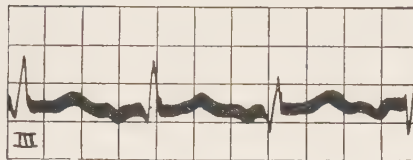
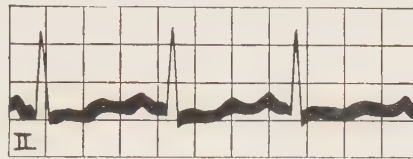
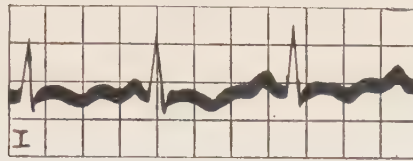


FIG. 309.—This record was obtained two days later (Fig. 308) and shows well marked inversion of the T waves in lead I and diphasic contours in lead II.**

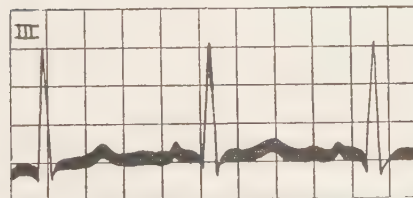
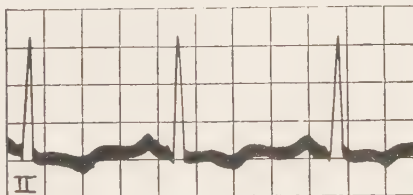
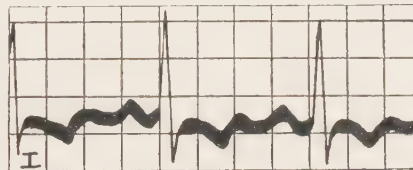


FIG. 310.—This record was obtained four days later (Fig. 309) on the day that left hemiplegia from cerebral infarction developed. The infarction was the result of a detached mural cardiac thrombus. Death occurred on this day, and necropsy revealed coronary thrombosis with infarction of the left ventricle and embolic infarction of the brain. The T waves in both leads I and II are markedly inverted.**

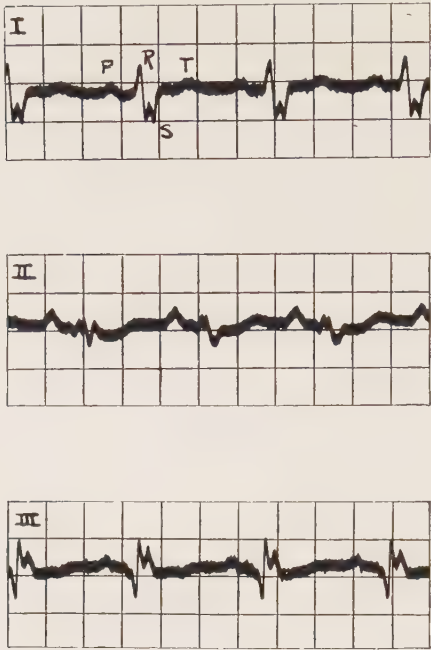


FIG. 311.—This record and the two following records were obtained from a man, aged fifty-two years, with coronary sclerosis. Two weeks before this record was obtained, the patient suffered from coronary thrombosis. The record shows the presence of incomplete bundle-branch block with diphasic T waves in lead III.

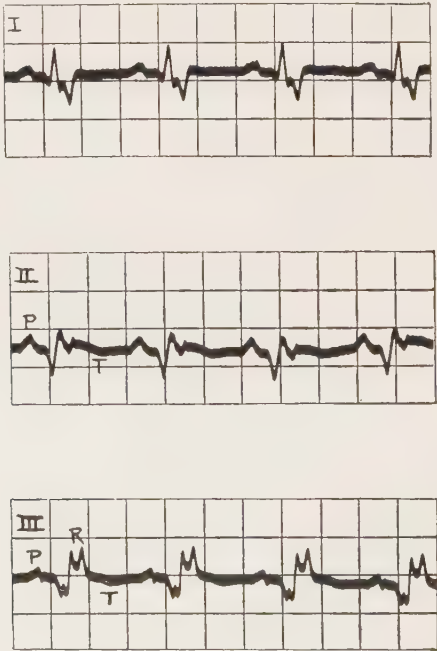


FIG. 312.—Record obtained seven days later (Fig. 311) and eighteen hours after the second attack of coronary thrombosis. The T waves in leads II and III are now inverted.

existing coronary sclerosis. The transitions following the occlusion are interesting (Figs. 307 to 310).

The fifth case represents two definite attacks of coronary closure which occurred within three weeks of each other (Figs. 311 to 313).

In order to avoid possible misunderstanding regarding these electrocardiographic changes I wish to emphasize that their occurrence was entirely independent of the action of drugs. The only drug administered to these patients was morphine sulphate.

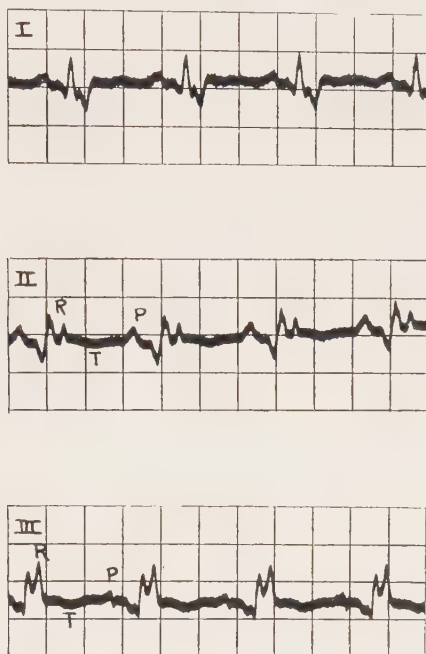


FIG. 313.—Record obtained the following day (Fig. 312) and on the day of the patient's death. It shows more marked inversion of the T waves in leads II and III. Necropsy verified the clinical diagnosis of two separate and distinct coronary occlusions with infarction of the myocardium.

The almost constant occurrence of T-wave negativity in coronary thrombosis is important evidence which tends to link this characteristic with obliterative coronary disease. In establishing the relationship, acute vascular occlusion must be ruled out.

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Chapter XVI

THE DYING HUMAN HEART

Unusual electrocardiograms are recorded during the period while death is taking place. In order to obtain such records, continuous observation is necessary from the time that death appears to be imminent until all galvanometric activity ceases. Individual variations in different cases occur but the general trend of the electrocardiograms in all cases

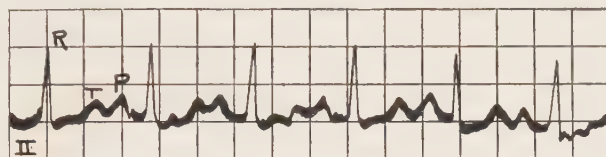


FIG. 314.—This record and those in the succeeding twenty-two figures were obtained just preceding death and during the time when death was actually taking place. All records are reproduced in lead II. The patient was a man, aged fifty-eight years, with subacute bacterial endocarditis (*Streptococcus viridans*). Almost continuous records were obtained but only representative transitions are reproduced here. This record, taken when death appeared imminent, shows no abnormalities.**

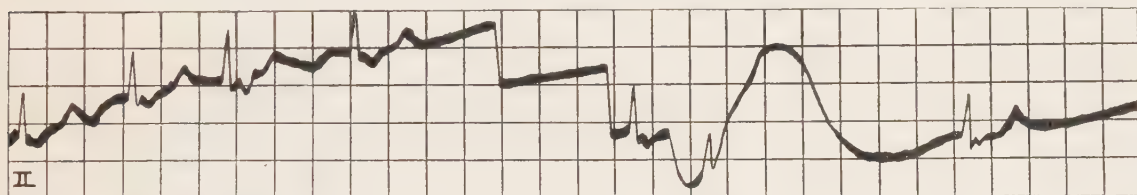


FIG. 315.—This record obtained forty-six minutes later (Fig. 314) shows the inception of nodal (A-V) rhythm with retrograde conduction. This record is somewhat deformed by a sudden movement of the patient.**

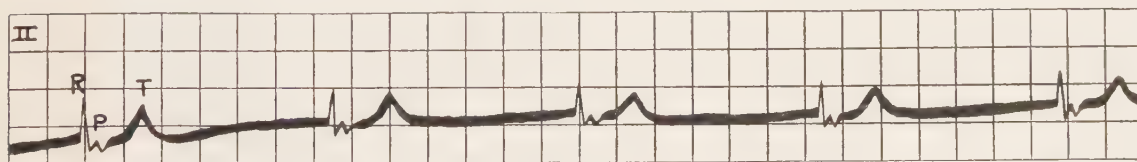


FIG. 316.—This record, obtained five seconds later (Fig. 315), shows slow nodal (A-V) rhythm with retrograde conduction. The rate is forty-six contractions each minute.

studied is similar. There is little difference between the records from patients with normal hearts and those from patients with advanced heart disease.

Certain definite characteristics appear in these electrocardiograms. In all the cases that I have observed, cessation of auricular activity preceded cessation of ventricular activity by an interval of from 14.8 seconds to 8.5 minutes. Cases have been reported in which the auricles were active after all ventricular activity had ceased. The various abnormal mechanisms that have been recorded are auricular fibrillation, auricular flutter, complete heart-block, delayed auriculoventricular conduction, partial heart-block, com-

plete bundle-branch block, sino-auricular block, ventricular fibrillation, nodal rhythm, migration of the pacemaker, and fusion of the R and the T waves.

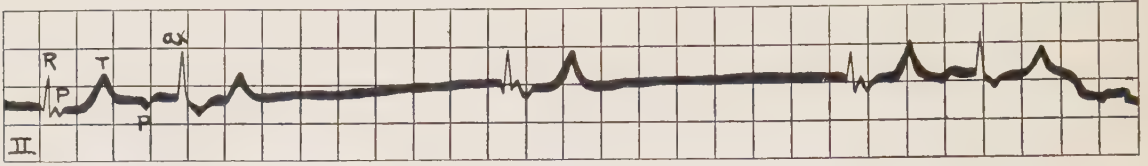


FIG. 317.—This record was obtained 42.8 seconds later (Fig. 316) and shows very slow nodal (A-V) rhythm with retrograde conduction interrupted by premature auricular contractions.**

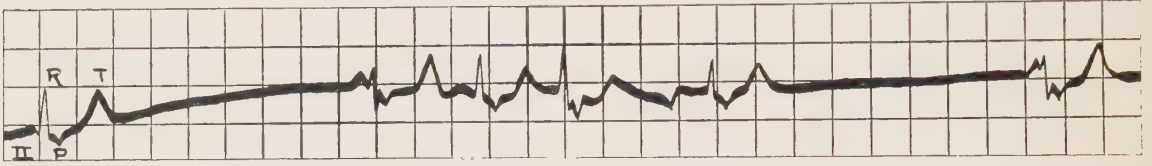


FIG. 318.—This record, obtained three minutes later (Fig. 317), shows great variations in rate, ranging from 33 to 100 contractions each minute. Nodal (A-V) rhythm with retrograde conduction still dominates.**

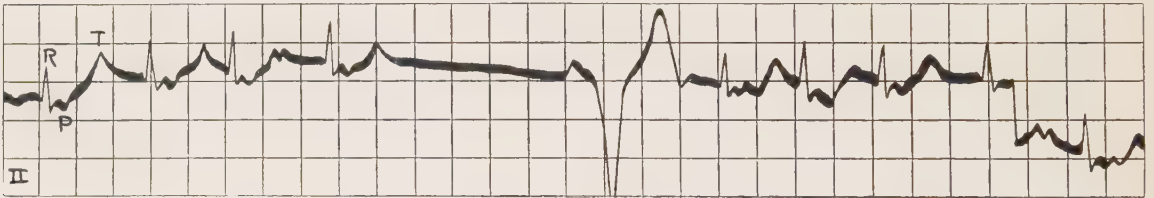


FIG. 319.—This record, obtained one minute later (Fig. 318), still shows nodal (A-V) rhythm with retrograde conduction as the dominant mechanism. One large, bizarre complex occurs which ultimately is shown to be caused by interference with conduction in a bundle branch.**

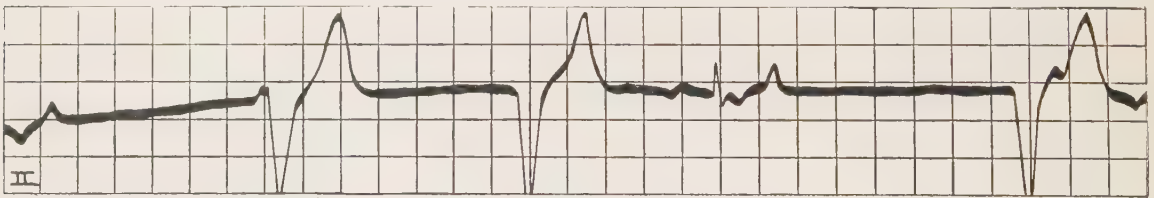


FIG. 320.—This record is continuous with the one in Figure 319 and shows further evidence of disturbance in bundle-branch conduction. Note the complexes of extremely high amplitude.**

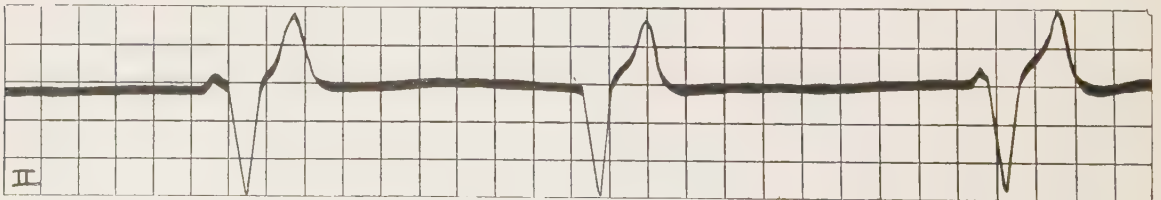


FIG. 321.—This record, obtained fifteen seconds later (Fig. 320), still shows disturbance in bundle-branch conduction.**

Records illustrative of the events which transpire in the dying human heart are shown in Figures 314 to 368. The reader is referred to the full, descriptive legends which give the time relationships of the records.

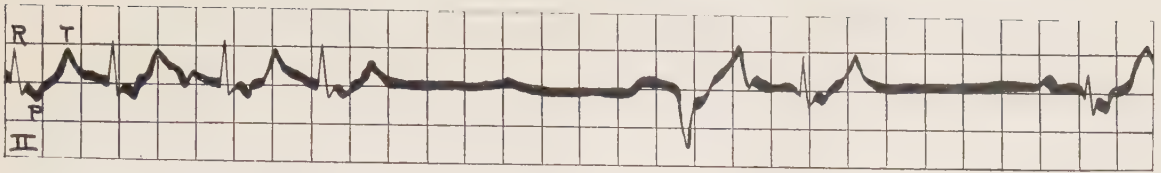


FIG. 322.—This record, obtained thirty seconds later (Fig. 321), shows the reestablishment of nodal (A-V) rhythm with retrograde conduction. A premature ventricular contraction occurs. Definite delay in auriculoventricular conduction occurs; the P-R interval is 0.28 second.**

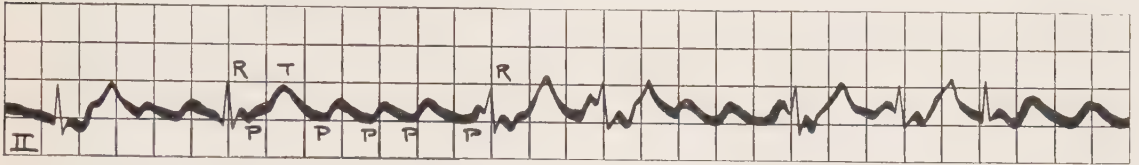


FIG. 323.—This record was taken thirty seconds later (Fig. 322) and shows the onset of auricular flutter. There is a variation in the degree of block present.**

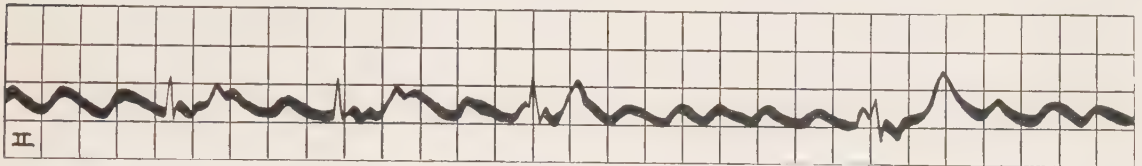


FIG. 324.—This record was obtained 10.2 seconds later (Fig. 323) and shows auricular flutter with increasing degrees of block.**

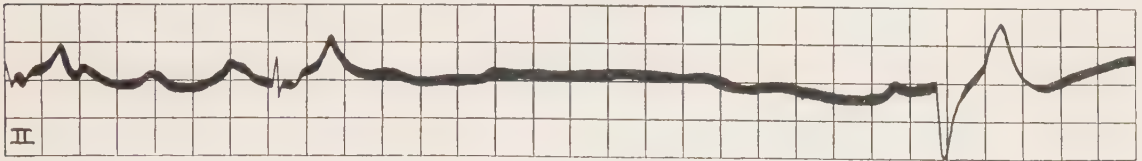


FIG. 325.—This record, obtained fourteen seconds later (Fig. 324), shows a long period of cardiac asystole followed by recovery occurring with a bizarre complex.**

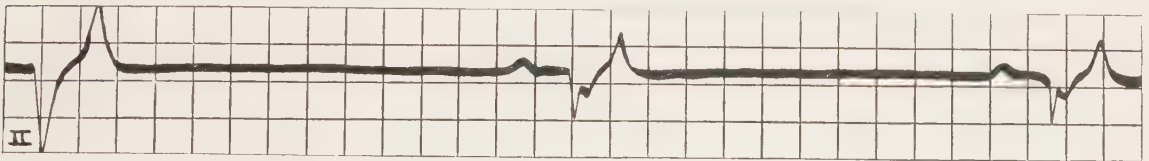


FIG. 326.—This record was obtained 5.4 seconds later (Fig. 325) and shows very slow cardiac action; the rate was only 25 contractions each minute. Aberrant ventricular complexes are present and there is marked delay in auriculoventricular conduction; the P-R interval is 0.32 second.**

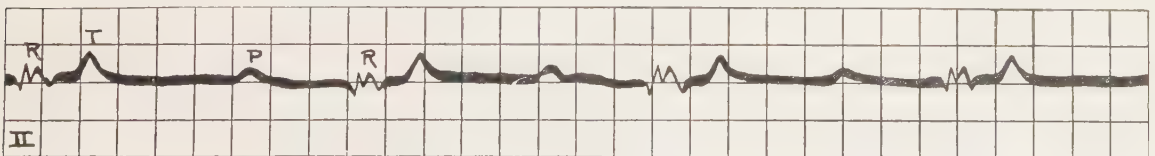


FIG. 327.—Record obtained ten seconds later (Fig. 326). It shows further delay in auriculoventricular conduction; the P-R intervals vary from 0.38 to 0.45 second. Note the change that has taken place in the ventricular complexes. They are now of very low amplitude and show deep notching.**

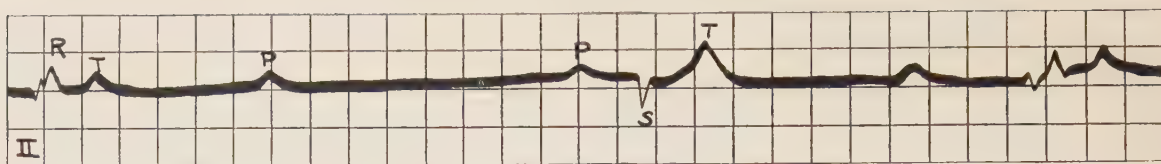


FIG. 328.—Record obtained one minute later (Fig. 327) showing complete heart-block. The ventricular rate is 21, the auricular rate is 33 contractions each minute.**

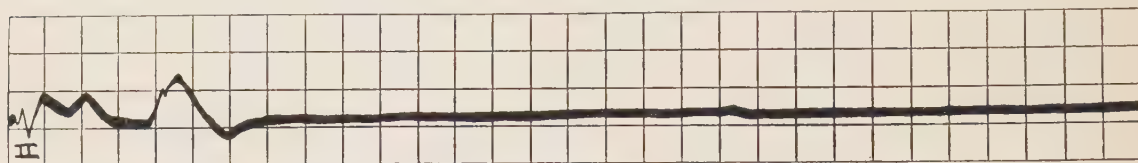


FIG. 329.—This record, obtained one minute later (Fig. 328), shows a long period of cardiac asystole.**

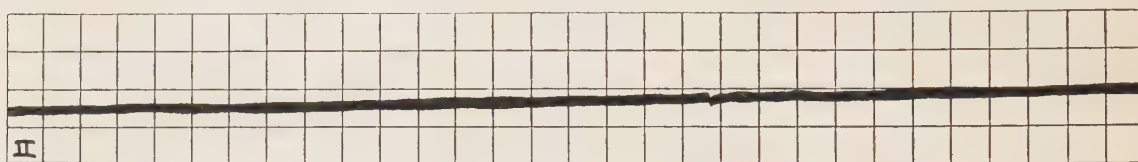


FIG. 330.—This record is continuous with the one in Figure 329. It still shows cardiac asystole which exists for a period of 11.4 seconds. The tiny deflection which appears in this strip is evidently an artifact.**

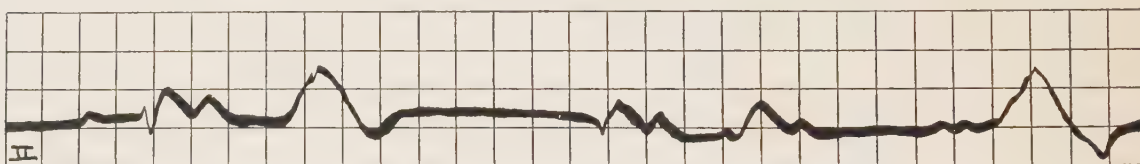


FIG. 331.—This record is continuous with the one in Figure 330 and shows the onset of ventricular fibrillation.**

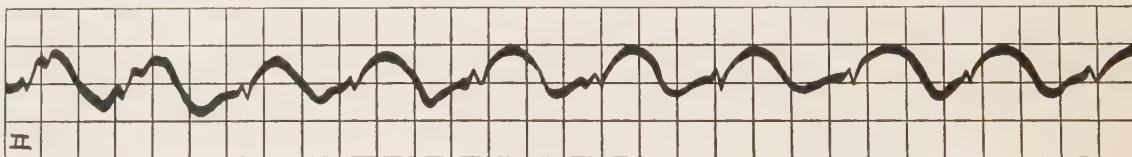


FIG. 332.—This record is continuous with the one in Figure 331 and shows some restoration of regularity. However, the rhythm is probably still that of ventricular fibrillation.**

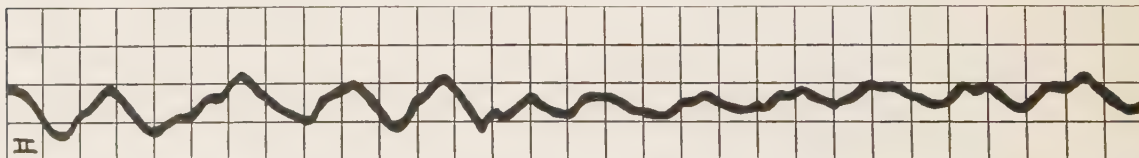


FIG. 333.—This record is continuous with the one in Figure 332 and shows ventricular fibrillation.**



FIG. 334.—This record was obtained ten seconds later (Fig. 333) and also shows ventricular fibrillation.**

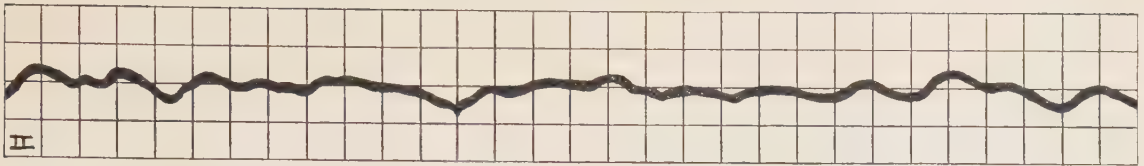


FIG. 335.—This record is continuous with the one in Figure 334 and shows ventricular fibrillation.**



FIG. 336.—This record is continuous with the one in Figure 335 and shows the termination of cardiac activity.**

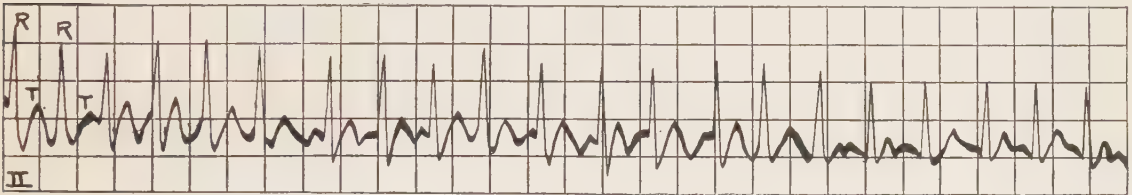


FIG. 337.—This record and those in the succeeding ten figures are in lead II and were obtained from a man, aged forty-three years, who was dying in the crisis of exophthalmic goiter. The records begin when death appeared imminent. Almost continuous tracings were obtained but they are too numerous to be reproduced in their entirety. Records showing important changes, with their time relations, are reproduced here. This record shows rapid auricular fibrillation; the rate is 222 contractions each minute.**

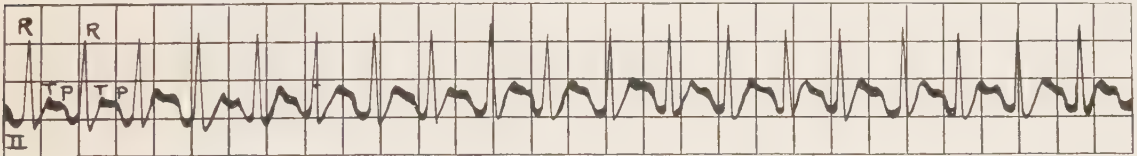


FIG. 338.—This record was obtained an hour and twenty-two minutes later (Fig. 337) and shows a regular tachycardia; the rate is 192 contractions for each minute.**

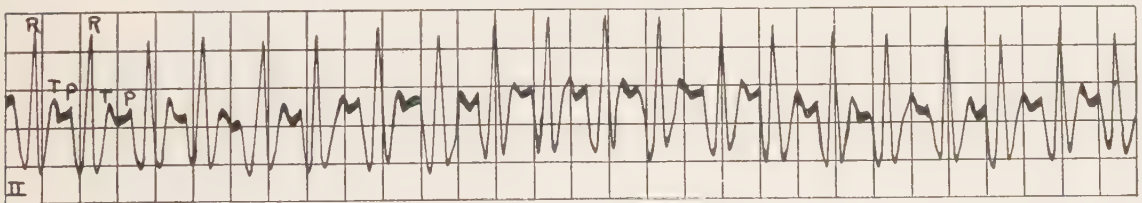


FIG. 339.—This record was obtained an hour and eight minutes after the record given in Figure 338 and shows regular tachycardia with a rate of 200 contractions each minute. Note the complexes of high amplitude.**

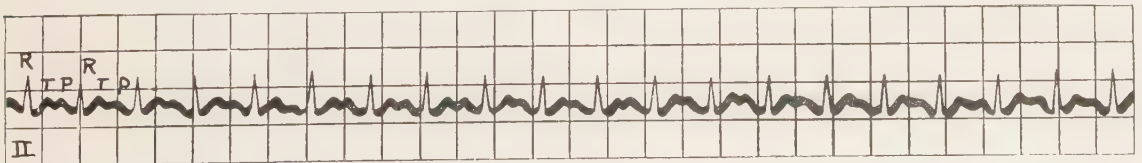


FIG. 340.—This record was obtained five minutes later (Fig. 339) and shows a rapid, regular tachycardia of 200 contractions each minute; complexes of very low amplitude.**

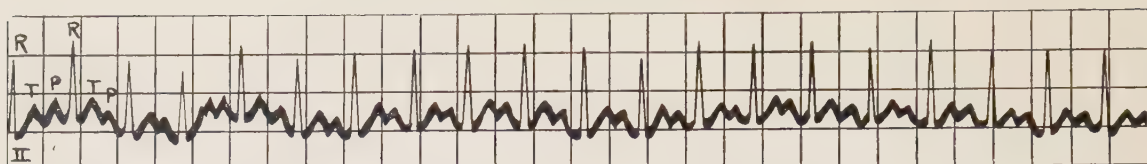


FIG. 341.—This record, obtained one hour and fifty-five minutes later (Fig. 340), still shows a rapid regular tachycardia of 200 contractions each minute. The complexes are of greater amplitude than those in the previous record.**

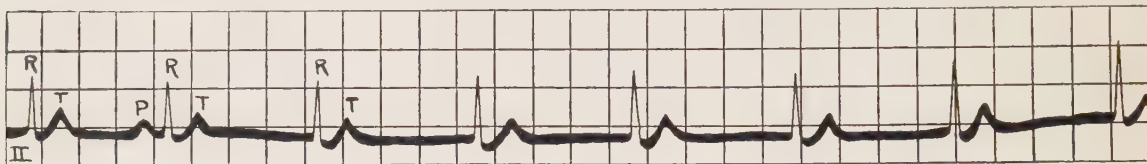


FIG. 342.—This record was obtained forty-five minutes later (Fig. 341) and shows the inception of nodal (A-V) rhythm with marked slowing in rate to 72 contractions each minute. The first complex in the record is in response to a sinus stimulus, as evidenced by a normal P wave. The P waves are absent from the remaining portion of the record.**

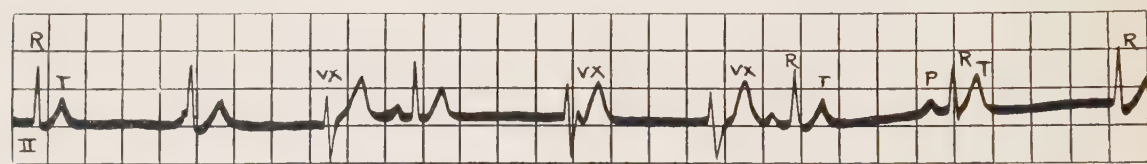


FIG. 343.—This record was obtained 41.4 seconds later (Fig. 342) and shows nodal (A-V) rhythm interrupted by occasional premature ventricular contractions and one normal complex.**

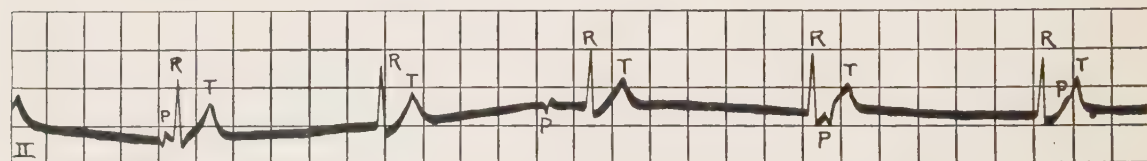


FIG. 344.—This record was obtained one minute later (Fig. 343) and shows a migratory pacemaker; the P waves continually bear different relationships to the ventricular complexes. The rate is the very slow one of 56 contractions each minute.**

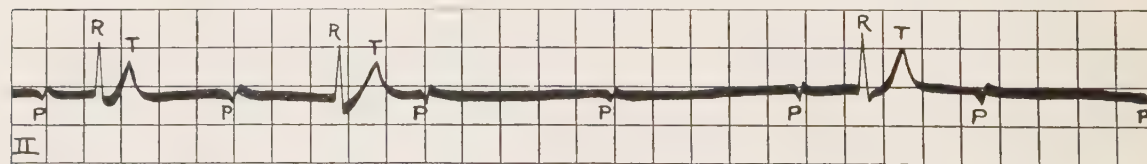


FIG. 345.—This record was obtained one second later (Fig. 344) and shows complete heart-block. Note the inversion of the P waves.**

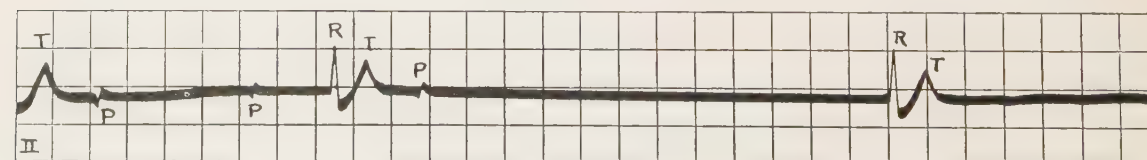


FIG. 346.—This record, taken eight seconds later (Fig. 345), shows the cessation of auricular activity. Note the persistently well defined ventricular complexes.**

A very striking observation in these electrocardiograms is the sudden onset of marked bradycardia with establishment of nodal rhythm. The events which transpire subsequent to the nodal rhythm appear to be variable, as illustrated in the accompanying figures. Ventricular fibrillation is frequently, but not always, the terminal mechanism.

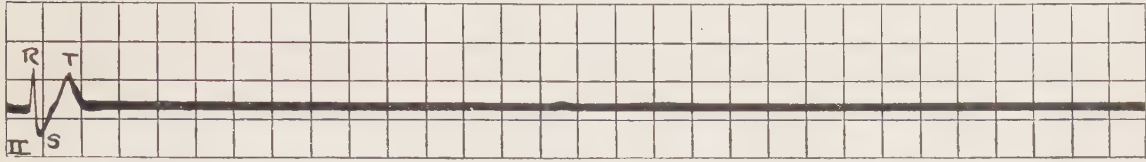


FIG. 347.—This record, taken 6.8 seconds later (Fig. 346), shows the final evidence of cardiac activity in the presence of a fairly normal QRS T complex. Beyond this point the record remained an iso-electric line.**

These data permit of interesting speculation as to their meaning and one concept may be summarized in the following manner: the fairly uniform onset of nodal rhythm, with marked slowing of the heart, and the varied phases of block that frequently are associated with these characteristics are suggestive of marked vagal action. These phenomena are

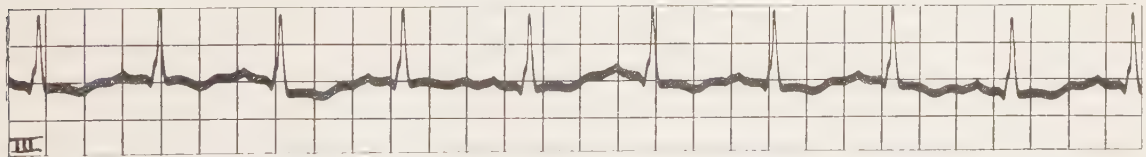
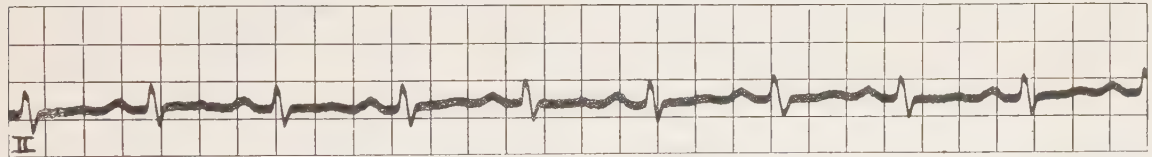
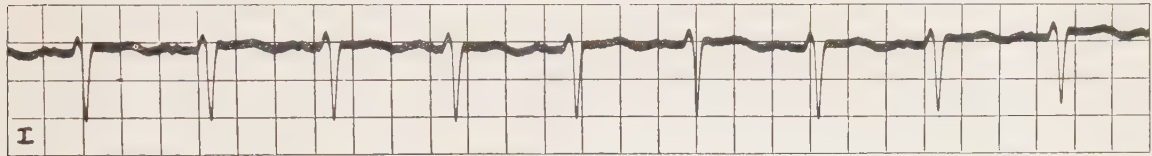


FIG. 348.—This record, and the succeeding records, were obtained before death and during the time when death actually was taking place. The patient was a woman, aged twenty-seven years, with severe essential hypertension and cardiac hypertrophy with congestive failure. This record, taken in all leads, shows rather low and poorly defined T waves in leads I and II. There is slight notching of the QRS complexes in lead III and marked preponderance of the right ventricle.

present at about the time that death appears to have occurred and when, undoubtedly, a marked degree of both cardiac and cerebral asphyxia is present.

Cerebral or cardiac asphyxia, or both, have been produced experimentally and graphic transitions have appeared which are similar to those that have been produced by the dying

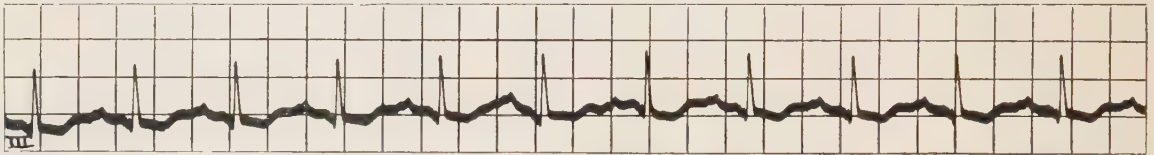
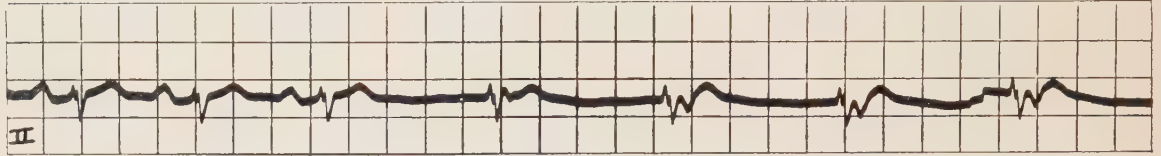
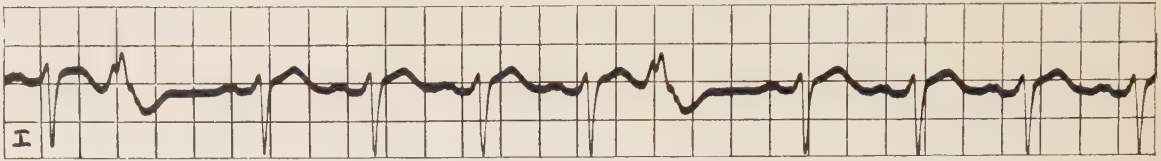


FIG. 349.—This record, obtained in all leads, was taken nineteen days later (Fig. 348) and one-half hour before death. Occasional premature ventricular contractions occur and there is considerable deformity of the QRS complexes in lead II.

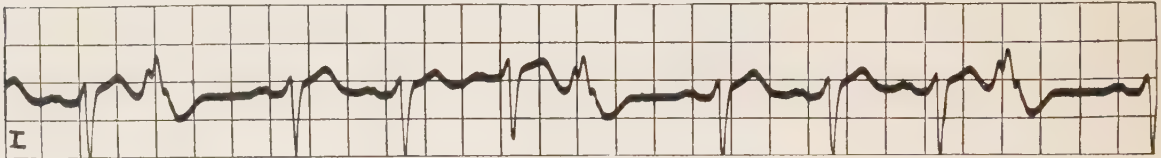


FIG. 350.—This record, in lead I, was obtained a few minutes later (Fig. 349) and is similar to the preceding figure.

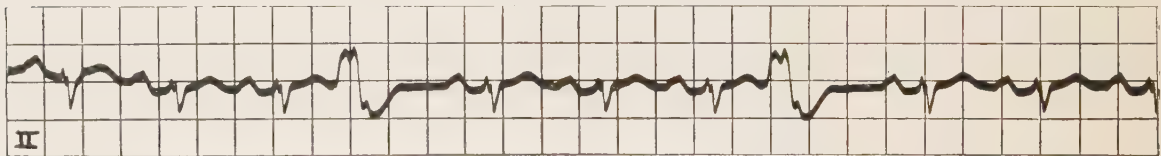


FIG. 351.—This record, in lead II, was obtained a few minutes later (Fig. 350) and shows occasional premature ventricular contractions.

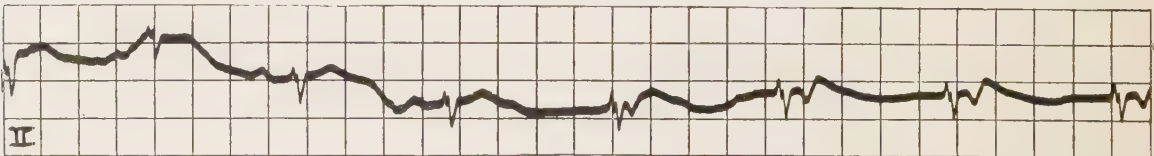


FIG. 352.—The record shown here, taken in lead II, is almost continuous with the record shown in Figure 351. Note in the latter portion of the strip the changes which apparently represent nodal rhythm with retrograde conduction.

human heart. The later changes observed, namely, long periods of complete cardiac asystole, the fusion of components of waves, and ventricular fibrillation may result from more profound changes which occur in the musculature itself. The latter changes are present a short time after clinical evidence of death has been noted.

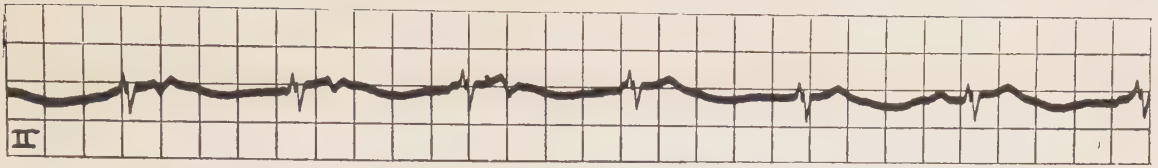


FIG. 353.—This record, continuous with the one given in Figure 352, shows the disappearance of nodal rhythm.

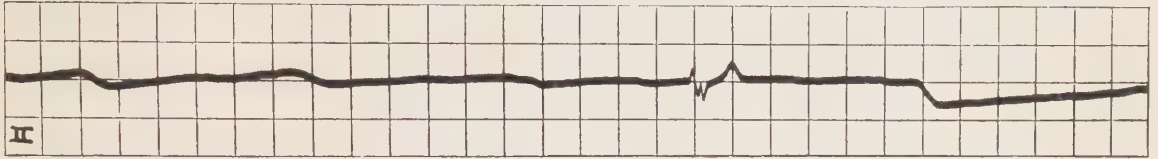


FIG. 354.—This record, in lead II, was taken a little later (Fig. 353) and shows long periods of cardiac asystole with no evidence of auricular activity.



FIG. 355.—Record in lead II continuous with that in Figure 354.

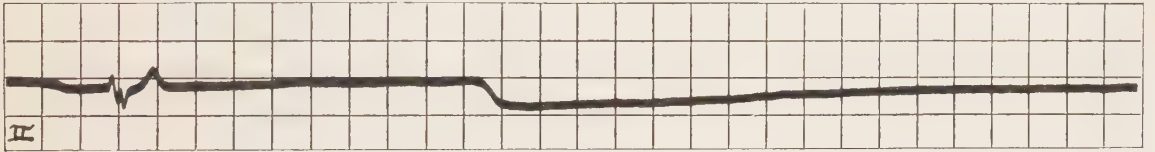


FIG. 356.—Record in lead II continuous with that in Figure 355. Only one ventricular complex is evident.

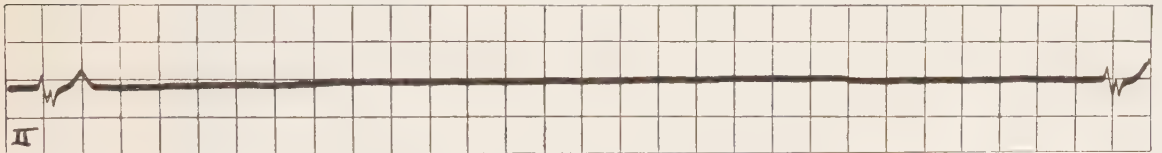


FIG. 357.—Record in lead II continuous with that in Figure 356.



FIG. 358.—Record in lead II continuous with that in Figure 357.

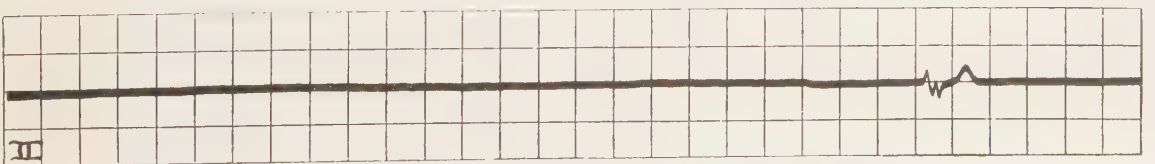


FIG. 359.—Record in lead II continuous with that in Figure 358.

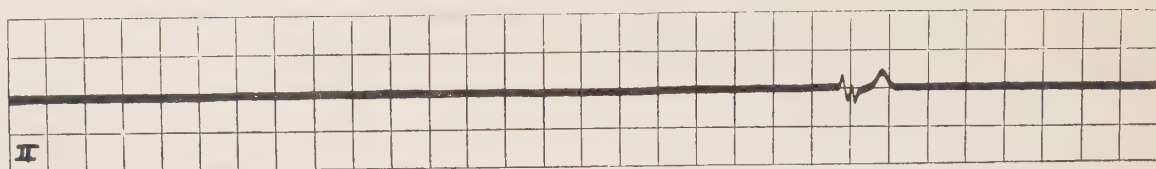


FIG. 360.—Record in lead II continuous with that in Figure 359.



FIG. 361.—Record in lead II continuous with that in Figure 360.

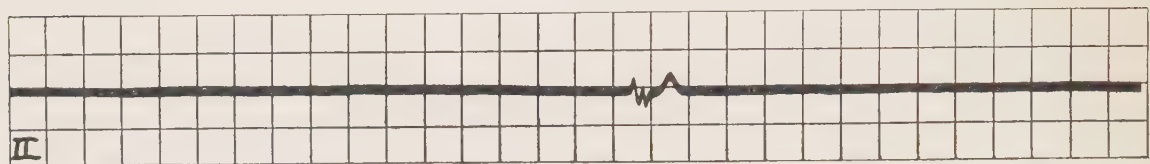


FIG. 362.—Record in lead II continuous with that in Figure 361.



FIG. 363.—Record in lead II continuous with that in Figure 362.

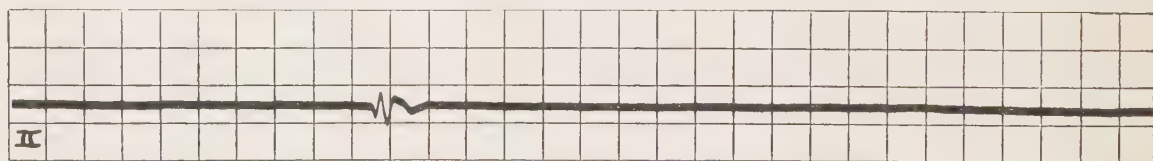


FIG. 364.—Record in lead II continuous with that in Figure 363.

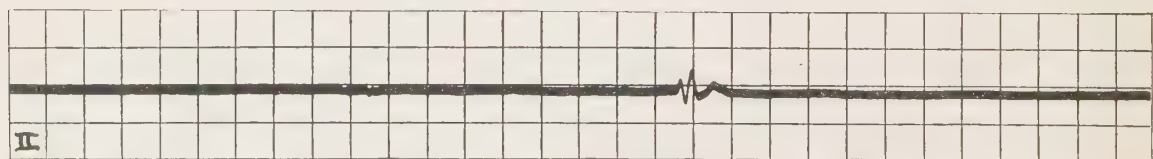


FIG. 365.—Record in lead II continuous with that in Figure 364.

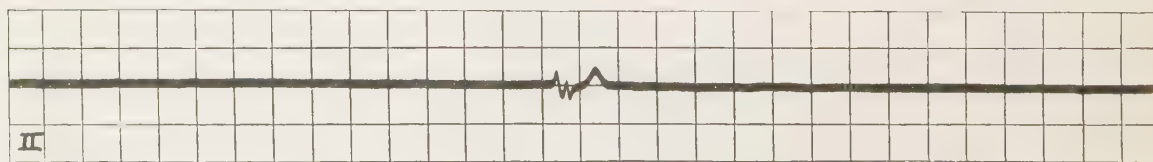


FIG. 366.—Record in lead II continuous with that in Figure 365.



FIG. 367.—Record in lead II continuous with that in Figure 366 and displaying the final complex.



FIG. 368.—Record in lead II continuous with that in Figure 367 and displaying no evidence of cardiac activity. This is an example of a case in which death occurs without the development of ventricular fibrillation.

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